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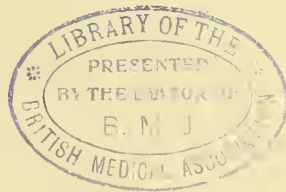
DISEASES

OF CHILDREN

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CANCELLED

AN AMERICAN TEXT-BOOK OF THE DISEASES OF CHILDREN.

INCLUDING

SPECIAL CHAPTERS ON ESSENTIAL SURGICAL SUBJECTS; ORTHOPÆDICS;
DISEASES OF THE EYE, EAR, NOSE, AND THROAT; DISEASES OF THE
SKIN; AND ON THE DIET, HYGIENE, AND GENERAL
MANAGEMENT OF CHILDREN.

BY AMERICAN TEACHERS.

EDITED BY

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PREFACE TO THE SECOND EDITION.

To keep up with the rapid advances in the field of pædiatrics and to round into a more perfect treatise the work so admirably accomplished by the various authors, most of whom labored entirely independently of one another, the whole subject matter embraced in the first edition of this work has been carefully revised; new articles have been added; some of the original papers have been emended, and a number have been entirely rewritten and brought up to date. For greater accuracy in classification, the section on the Infectious Diseases has been rearranged so as to embrace Tuberculosis and Malaria. The new articles include "Modified Milk and Percentage Milk Mixtures," "Lithæmia," and a section on Orthopædics; those rewritten are "Typhoid Fever," "Rubella," "Chicken-pox," "Tuberculous Meningitis," "Hydrocephalus," and "Scurvy"; while more or less extensive revision has been made in the chapters on Infant Feeding, Measles, Diphtheria, and Cretinism. The volume has been thus increased in size by fully fifty pages of fresh material.

The editor records with profound regret the decease of two of his most valued collaborators—Dr. Charles Warrington Earle, of Chicago, and Dr. J. Lewis Smith, of New York—to whose pioneer work in pædiatrics the medical profession owes a lasting debt of gratitude.

The editor gratefully acknowledges the flattering reception accorded the first edition of the work, and expresses his thanks to Dr. Thompson S. Westcott for his most efficient assistance in the preparation of the revision.

LOUIS STARR.



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PREFACE.

IN the preparation of this volume the Editor's object has not been to add unnecessarily to the number of encyclopædias already existing, but to present to the profession a working text-book which shall be closely limited to, while completely covering, the field of pediatrics.

To make such a book useful to the practitioner, who must too often read as he runs, and to the student, who of necessity is unable to devote his study hours to one branch of medical science, but must divide them between many general and special subjects, it seems essential that certain conditions should be closely adhered to. These are—first, careful condensation, without omission, that the whole subject may be embraced between the covers of one readily handled volume; second, limitation of the subject-matter to such practical points as Etiology, Symptomatology, Diagnosis, and Treatment including Feeding, Hygiene, Therapeutics and the Prevention of Disease, while avoiding, so far as possible, the insertion of references to journals or authorities, of more interest to those engaged in research than to those in active practice; third, the selection of a large staff of collaborators from the most important medical centres of our country, to secure for each subject the care of the authority best fitted to portray it, to give the work broadness and stamp it with a national, rather than a sectional, imprint; fourth, so to time the publication that, without undue haste, each article contributed should have the same freshness, and the book as a whole be thoroughly abreast with the rapid advance which is constantly made in this branch of our profession; finally, the addition of chapters upon certain subjects which, though usually treated specially and separately, constantly come under the notice of those who work with, or study, the ills of childhood, such as diseases of the eye, the ear, the skin, the nose and throat, and the anus and rectum; circumcision, tracheotomy, intubation, vesical calculus, venereal disease and allied subjects. These conditions we have endeavored to fulfil.

In conclusion, the Editor desires to thank individually the collaborators he has been so very fortunate in securing, and to tender them, in advance, the greater share of whatever credit may attend the venture. His thanks are also due to Dr. Thompson S. Westcott for his most efficient and interested assistance.

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AN AMERICAN TEXT-BOOK OF THE DISEASES OF CHILDREN.

INTRODUCTION.

THE CLINICAL INVESTIGATION OF DISEASE AND THE GENERAL MANAGEMENT OF CHILDREN.

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I. THE CLINICAL INVESTIGATION OF DISEASE.

EARLY life may be divided into two periods—namely, infancy and childhood. Infancy is the time elapsing between birth and the complete eruption of the milk teeth, an event that transpires about the end of the second year of life; childhood extends from this age to the development of puberty, about the age of thirteen or fifteen years.

Of the diseases that may occur during these periods a few are peculiar to the time of life, or are “children’s diseases” proper; others, while identical in class with the ordinary affections of adult and mature years, are variously modified in symptoms and course by conditions inherent to early age; but in all the clinical investigation is beset with difficulties which the student must be prepared to overcome. Thus, the absence of speech in the infant deprives us of the important assistance afforded by correctly described subjective symptoms, and renders it necessary to look to the mother or nurse for the history of an illness. In older children the case is little better, since with them words are not prompted by sufficient knowledge to be of great service. Further, the wilfulness, dislikes, fear, and agitation of the child are impediments which must be overcome before a satisfactory examination can be made, and which will often tax the skill and patience of the physician to the utmost in the overcoming. Another source of difficulty lies in the activity of growth and development in infants, which renders them liable to be affected by slight causes, and makes disease sudden in its attack, short in its course, and intense in its symptoms. The rapid development of the nervous system especially leads to confusion. The nerves bind every portion of the frame in a sympathy so close that an affection of a single part may cause marked general disturbance, and local symptoms are often reflected, directing attention to organs very distant from those really diseased. Finally, the extreme excitability of the nervous system of healthy children often causes a trifling illness to assume an aspect of the greatest gravity; while, on the contrary, the depression of nervous sensi-

bility that attends chronic wasting diseases so obscures the symptoms that a dangerous intercurrent affection may appear trifling or remain altogether latent.

On the other hand, to offset these difficulties, disease in the child is usually uncomplicated, rarely has its course and symptoms modified by tissue lesions the result of previous affections, and never by vicious habits, such as the abuse of stimulants and narcotics, or by mental overwork and nerve-strain. The confusing element of misstated subjective symptoms is also absent, while correct diagnosis is greatly aided by the facility with which physical examination of the whole body may be practised.

In conducting the investigation it is well to proceed in three regular stages, as follows: 1st. Questioning the attendants; 2d. Inspecting the child; 3d. Physical examination.

1. QUESTIONING THE ATTENDANTS.

When the patient is under eight or ten years of age, the only way of obtaining a knowledge of the previous history and of what may occur between visits is carefully to question the mother or nurse. The account must be patiently elicited, and credited with due reference to the narrator's intelligence. It is well never entirely to discredit a statement without good reason, for many women, though weak and foolish in other respects, are excellent observers when their powers are guided by affection. Besides, being thoroughly acquainted with their children's habits and dispositions, they will often detect deviations from health that the physician might overlook entirely. This part of the examination, particularly when the acquaintance and good-will of the child have not previously been obtained, should, if possible, be made before entering the sick-room.

As there are certain points about which it is always necessary to be informed, the adoption of a definite order of questioning is advisable.

The family history as far back as the parents should first be ascertained, inquiry being chiefly directed to the detection of chronic maladies and transmissible diseases, as tuberculosis and syphilis. If any deaths have occurred, their causation should be investigated; and an inquiry into the occurrence, or the reverse, of previous stillbirths is often important. Then an outline of the child's life from birth up to the date of the illness in question must be obtained. This should include the following items: The manner of feeding during infancy—whether at the breast or from a bottle, and if the latter, the composition of the food employed; the date of commencement and the regularity of dentition; the general state of health in regard to strength or weakness and liability to illness; the time of occurrence and the nature of any prominent attack of illness, especially of the eruptive fevers; whether vaccination has been performed or no; the hygienic surroundings—for instance, the healthfulness of the locality of residence, the sort of house and room occupied, and the character of the clothing and food. In older children, if at school, the time devoted to study, and if at labor, the nature and the hours of work.

After this it is necessary to fix the time the attack in hand began. The occurrence of some striking symptom, as convulsions or violent vomiting, often establishes this point beyond a doubt; but when there is any uncertainty the best plan is to question back, day by day, until a time is reached at which the child was perfectly well, and to date the onset from this period. The most common of the general indications of commencing illness are disturbed sleep and irritability of temper.

The next step is to learn the mode of attack and the symptoms and course of the disease prior to the first visit. The questions now must be general, never leading. They must be sufficiently exhaustive to touch upon all the functions of the body, and when a trail is started it must be patiently followed to the end. Alterations in sleep, bodily strength, surface temperature, appetite, digestion, urine elimination, respiration, and so on, must be sought for, and the account of such deviations from the normal state as vomiting, diarrhœa, or cough will suggest further questions, as well as point out the path to be followed in the future examination.

This portion of the investigation is closed by an inquiry into the treatment that may have been already adopted.

2. INSPECTING THE CHILD.

When the eye and ear of the physician are trained to their work, valuable information can be obtained by simply looking at an ill child and listening to its cry or spoken words. Even while the child is lying asleep or sitting quietly in the nurse's lap many facts may be learned; but this portion of the examination is never complete without an inspection of the naked body. The points thus ascertained consist in alterations in the expression of the face, in decubitus, in the appearances of the body, and so on, and may be designated the *features* of disease. The relative position of the observer and patient during inspection is of importance. If possible, the former should stand with his back to, and the latter be so placed that his face is toward, a window or lamp. The light must never be strong enough to dazzle when the countenance is the object of inspection, as this causes distortion of the features.

For convenience, the *features* of disease will be studied under different headings; and since to appreciate them it is necessary to have a knowledge of the healthy aspect, both the normal and abnormal appearances will be described.

FACE.—The face of a healthy sleeping child wears an expression of perfect repose. The eyelids are completely closed, the lips slightly parted, and while a faint sound of regular breathing may be heard, there is no perceptible movement of the nostrils. Incomplete closure of the lids, with more or less exposure of the whites of the eyes, is noted when sleep is rendered unsound by moderate pain and during the course of all acute and chronic diseases, particularly when they assume a grave type. Twitching of the lids heralds the approach of a convulsion, and at such times, too, there is often oscillation of the eyeballs or squinting. A marked smile, due to contraction of the muscles about the mouth, signifies abdominal pain or colic, and pursing out of the lips and chewing motions of the jaw, gastro-intestinal irritation. Dilatation of the *alæ nasi*, with or without noisy breathing, points to embarrassed respiration, the result of extensive bronchial catarrh, pneumonia, or pleurisy with effusion.

When awake and passive the healthy infant's face has a look of wondering observation of whatever is going on about it. As age advances the expression of intelligence increases, and every one is familiar with the bright, round, happy face of perfect childhood, so indicative of careless contentment and so mobile in response to emotions.

The picture is altered by the onset of any illness, the change being in proportion to the severity of the attack. An expression of anxiety or of suffering appears, or the features become pinched and lines are seen about the eyes and mouth. Pain most of all sets its mark upon the countenance, and by noting the feature affected it is often possible to fix the seat of serious disease. Thus, contraction of the brows denotes pain in the head; sharpness of the nostrils,

pain in the chest; and a drawing of the upper lip, pain in the abdomen. As a rule, the upper third of the face is modified in expression in affections of the brain, the middle third in diseases of the chest, and the lower third in lesions of the abdominal viscera. Puffiness of the eyelids and a fulness of the bridge of the nose indicate dropsy, and should direct attention to the kidneys. When there is a tuberculous tendency the face is often oval, the features delicate, and the expression intelligent; the hair fine and silky; the skin smooth and transparent; the temporal veins visible; the eyelashes long and curving, the irides large and deep-colored, and the sclerotics pearly white or bluish; finally, a growth of fine hair is often noticeable on the temples and in front of the ears. On the contrary, the face may be round and heavy; the complexion doughy; the upper lip swollen; the nostrils wide and the alæ of the nose thick; the eyelids swollen and reddened at their edges; the hair coarse; and the lymphatic glands of the neck enlarged.

A marked disfigurement of the face may indicate one of several diseases, according to its character. For example, broadness or complete flatness of the bridge of the nose is significant of constitutional syphilis. A large, square head and projecting forehead, with a face of natural size or smaller, show that the child has suffered from rickets. An immense globular head, overhanging forehead, and diminutive face, with eyeballs projected downward and irides almost concealed by the lower lids, are pathognomonic signs of chronic hydrocephalus.

DECUBITUS.—The complete repose depicted on the countenance of a healthy sleeping child is shown also by the posture of the body. The head lies easy on the pillow; the trunk rests on the side, slightly inclined backward; the limbs assume various but always most graceful attitudes, and no movement is observable but the gentle rise and fall of the abdomen in respiration. In the waking state the child, after early infancy, is rarely still. The movements of the arms, at first awkward, soon become full of purpose as he reaches to handle and examine various objects about him. The legs are idle longer, though these, too, soon begin to be moved about with method, feeling the ground in preparation for creeping and walking.

With the onset of disease the scene changes. In acute attacks attended with pain sleep is no longer restful. The infant is content only when rocked, fondled, or "walked" in the nurse's arms. The older child tosses about uneasily in bed, or demands a constant change from the bed to the lap. During the waking hours the movements are purposeless, quick, and impatient, the position is constantly shifted, and frequent whining complaints are made. As a contrast to this condition of jactitation, at the beginning of the specific fevers children often lie quiet and drowsy for hours. In chronic affections attended with debility the movements become slow and languid, and in stupor and coma there are perfect stillness and immobility.

There are certain positions and gestures which have especial significance. Sleeping with the head thrown back and the mouth open is a frequent accompaniment of chronic enlargement of the tonsils. A tendency to "sleep high"—that is, with the head and shoulders elevated by the pillow—indicates impaired pulmonary or cardiac function. So, too, does an upright position in the nurse's arms, with the chest against her breast and the head hanging over her shoulder—a posture assumed by young children. "Sleeping cool"—namely, resting only after all the bed-clothing has been kicked off—is an early symptom of rickets. The position termed *en chien de fusil* is a symptom of the advanced stages of cerebral disease, especially tubercular meningitis. The child lies upon one side, with the head stretched far back, the arms pressed close to the

sides and folded across the chest, the thighs drawn up toward the abdomen, the legs flexed on the thighs, and the feet crossed. Restless movements of the head or boring of the head into the pillow also point to cerebral disease. A retained position, as on the back or one side, together with short, quick breathing, points to some inflammatory change in the respiratory or abdominal organs. Persistent lying on the face is an evidence of photophobia.

Of gestures, the frequent carrying of the hand to the head, ear, or mouth indicates headache, earache, or the pain of dentition respectively, and constant rubbing of the nose is a feature of gastro-intestinal irritation.

If the thumbs be drawn into the palms of the hands and the fingers tightly clasped over them, or if the toes be strongly flexed or extended, a convulsion may be expected. The presence of clonic contractions of the muscles, with unconsciousness, indicates, of course, a convulsion; while irregular, badly co-ordinated, jerky movements—consciousness being retained—attend chorea. In infants the existence of colic is shown by repeated extension and retraction of the legs, clenching of the hands into fists, flexion and extension of the forearms, and a writhing movement of the trunk. The fact of one limb remaining passive while the others are actively moved about naturally suggests motor paralysis.

THE SKIN.—In the new-born infant the color of the skin varies from a deep to a light shade of red. After the lapse of a week this redness fades away, leaving the surface yellowish-white, and in a fortnight the skin assumes its typical appearance. Allowing for natural variations in complexion, the skin of a healthy child is beautifully white, transparent, and velvety. The cheeks, palms of the hands, and soles of the feet have a delicate pink color, and the general surface is rosy in a warm atmosphere, marbled with faint blue spots or lines in a cool one. As age advances the coloring becomes more pronounced, and until the completion of childhood the complexion is much fresher than in adult life.

Lividity of the eyelids and lips is a sign of imperfect aëration of the blood and points to pulmonary or cardiac disease. Marked blueness of the whole face is a symptom of *morbus cæruleus*, and indicates a congenital malformation of the heart. On the other hand, a faint purple tint of the eyelids and around the mouth shows weak circulation merely, or, more frequently, deranged digestion. A decided yellow hue of the skin and conjunctivæ is seen in jaundice; an earthy tinge of the face in chronic intestinal diseases; a waxy palor in renal diseases; and paleness in any affection attended with exhaustion. Brownish-yellow discoloration of the forehead is significant of inherited syphilis; a bright, circumscribed flush on one or both cheeks, of inflammation of the lungs or pleura or of gastro-intestinal catarrh, according to its occurrence with or without an elevated temperature.

In addition to the cutaneous lesions of the eruptive fevers, each having its special characteristics, an eruption of herpetic vesicles on the lips may be mentioned as present both in pneumonia and in malarial fevers.

Slight want of proper aëration of the blood is shown by blueness of the finger-nails; a greater degree, by cyanosis of the whole hand. Deformity of the nails is a symptom of syphilis; clubbing of the finger-tips, of chronic lung disease; and redness, swelling, and suppuration about the nails, of struma. The dropsy of scarlatinal nephritis causes a puffiness and cushiony appearance of the dorsum of the hands. Often, too, in this condition, the finger-ends are glossy as if smeared with oil, and there is an exfoliation of the epidermis about the nails. The last two symptoms frequently serve to confirm a retrospective diagnosis of scarlet fever.

MODE OF DRINKING.—By watching an infant taking the breast or bottle some knowledge can be obtained of the condition both of the mouth and throat and of the respiratory organs.

If there be any soreness of the mouth, the nipple is held only for a moment, and then dropped with a cry of pain. When the throat is affected, deglutition is performed in a gulping manner, an expression of pain passes over the face, and no more efforts are made than required to satisfy the first pangs of hunger. Under similar circumstances older children drink little and refuse solid food entirely. An infant suffering from the oppression of pneumonia or severe bronchitis seizes the nipple with avidity, swallows quickly several times, and then pauses for breath. In older patients the act of drinking, which should be continuous, is interrupted in the same way.

If the finger be put into the mouth of a healthy baby, it will be vigorously sucked for some little time. The diminution of the act of suction during a severe illness is a sign of danger; its re-establishment a good omen. In conditions of stupor and coma it is noticeably absent.

THE CRY.—Crying is the chief, if not the only, means that the young infant possesses of indicating his displeasure, discomfort, or suffering. Even long after the powers of speech have been developed, the cry continues to be the main channel of complaint. It may be accepted as a rule that a healthy child rarely cries. Of course, some acute pain, as from a fall or accident or blow, will cause crying in the most healthy child, but the storm is quickly over. Incessant, unappeasable crying is due to one of two causes—namely, earache or hunger—and the distinction may readily be made by putting the child to the breast or offering a properly-prepared bottle. The *hydrencephalic* cry, denoting pain in the head, is a sudden, sharp, very loud, and paroxysmal shriek. Crying during an attack of coughing or for a brief time afterward, and attended with distortion of the features, indicates pneumonia. In acute pleuritis the cry also accompanies the cough, but it is produced too by movements of the body and by pressure on the affected side. It is louder, indicative of greater suffering, and sometimes most difficult to check. Intestinal pain causes crying just before or after an evacuation of the bowels, and is associated with wriggling movements of the body and pelvis and with eructation or the passage of flatus. Conditions of general distress or malaise predispose to fits of fretful crying, the paroxysms being excited by any disturbing influence, or even by merely looking at the little sufferer.

When the cry has a nasal tone, it indicates swelling of the mucous membrane of the nares or other obstructing condition. Thickening and indistinctness occur with pharyngeal affections. A loud, brazen cry is a precursor of spasmodic croup. Hoarseness points to a lesion of the laryngeal mucous membrane, either catarrhal or syphilitic in nature. In membranous croup and in some cases of extreme exhaustion the cry is faint and inaudible. Finally, in severe croupous pneumonia, in extensive pleural effusion, and in rickets ordinary disturbing causes are inoperative for the production of fits of crying, and there is a seeming unwillingness to cry, on account of the action interfering with the respiratory function.

The conditions of altered tone apply equally to the articulate voice in children who are old enough to speak.

The cough, too, must not be disregarded. Many of its characters correspond with the voice and cry. It is brazen in spasmodic croup, suppressed in true croup, hoarse in laryngeal catarrh, and so on. But it has certain features of its own. In bronchitis it is more or less paroxysmal, evidently dry in the early stages, loose and rattling as the catarrh "breaks up." In the

painful pulmonary affections, pneumonia and pleurisy, it is choked back, and whenever it occurs an expression of pain passes like a cloud over the face. In pertussis the peculiar spasmodic cough is the pathognomonic symptom. Cough is always unproductive—that is, unattended by expectoration—in children under seven years of age.

The formation of tears rarely begins before the third or fourth month of life. Subsequently, an alteration in this secretion may be of aid in forecasting the result of disease. The prognosis is bad when the tears become suppressed; good when the secretion continues during an illness or when it reappears after being suppressed.

There are several other sources of information which should be investigated before proceeding to the physical examination, although, strictly speaking, they do not come under the head of inspection of the child. These are the alterations in the odor of the breath, and the characters of the fecal evacuations, of the urine, and of material ejected by vomiting.

THE BREATH.—The breath of a healthy child is odorless, or, as the nurse will say, “sweet,” except perhaps immediately after taking nourishment, when it may, for a short time, have the smell of milk or other food. Any persistent odor is abnormal.

Any morbid condition of the system that prevents the elimination of metamorphosed nitrogenous tissue through the mucous membrane of the intestines or retards the passage of decomposing detritus along the bowels will cause an offensive breath. Under this head are conditions characterized by high temperature, catarrhal inflammation of the gastro-intestinal tract, chronic debilitating diseases, etc. The same result also frequently attends structural lesions of the kidneys. The reason for this is, that the system, in order to get rid of poisonous matter—for accumulated waste is poison—and to maintain the balance between the constant construction and destruction of tissue, must throw off elsewhere what the intestinal glands and the kidneys fail to excrete; so the lungs take on vicarious activity and the expired air becomes tainted. Purely local causes of *halitosis* also exist. These are decayed teeth, caries of the nasal and maxillary bones, ulceration of the mucous membrane of the mouth, nose, larynx, trachea, and bronchial tubes, and gangrene of the cheeks. Chronic poisoning by lead, arsenic, or mercury, though not very common in childhood, is another cause of ill-smelling breath.

To speak in general terms, the breath may become sour, catarrhal, foetid, gangrenous, ammoniacal, and stercoraceous. Sour breath is present, in infants more especially, when there is gastric fermentation. Catarrhal breath has numerous shades of difference. In chronic catarrh of the pharynx there is a “heavy” odor, not noticeable far from the patient’s face. It is always most marked during and after sleep. Should there be associated follicular tonsillitis, the breath, while still heavy, becomes extremely offensive, with a scent somewhat like that of decaying cheese, and is very penetrating. This odor, too, is worse after sleeping. At the onset of acute catarrh of the stomach the breath sometimes has a vinous odor, at others it is sweetish, and again it has the same quality as after an inhalation of ether. Later in the attack it becomes sour or has the odor of sulphuretted hydrogen. What is known as a “feverish breath” has a heavy, sweetish smell. It is met with in diseases of high temperature; thus, it is very marked and rapid in appearance in scarlatina.

Fœtor of the breath is observed in its mildest form in such affections as aphthæ and ulcerative stomatitis. It is better developed in ozæna and necrosis of the maxillary bones. Decaying teeth give much the same odor, though it is less strong and penetrating.

Noma gives rise to a gangrenous odor, and a patient so affected will fill the room in which he lies, or even a whole dwelling, with the most sickening stench. Cases of empyema, with ulceration of the lung and discharge of pus through the bronchial tubes, have an almost equally offensive breath, but here there is often a superadded flavor of garlic.

Ammoniacal breath is observed only in patients suffering with uræmic poisoning. A purely stercoraceous breath is rare, and when met with is an accompaniment of fecal tumor or of intussusception.

The different metallic poisons give rise to no characteristic odor, and it is necessary to look to the clinical history to determine the special poison.

THE FÆCAL EVACUATIONS.—The daily number of evacuations natural for a child varies greatly with its age. For the first six weeks there should be three or four stools every twenty-four hours. After this time, up to the end of the second year, two movements a day is the normal average. Subsequently, the frequency of defecation is usually the same as in adults—once per diem. During the first period the stools have the consistence of thick soup, are yellowish-white or orange-yellow in color, with sometimes a tinge of green, have a faint fecal, slightly sour odor, and are acid in reaction. In the second they are mushy or imperfectly formed, of uniform consistence throughout, brownish-yellow in color, and have a more fecal odor. The last two characters become more marked as additions are made to the diet. After the completion of the first dentition the motions have the same appearance as in adult life; they are *formed*, and brownish in color, with a decided fecal odor.

Many alterations occur in disease. The frequency of the movements may be increased, constituting diarrhœa, or lessened, constituting constipation. In the former condition the consistency is diminished, in the latter increased. Instead of being uniform throughout, the stool may be mixed, partly liquid, partly solid, indicating imperfect digestion, and curds of milk and pieces of undigested solid food may be mingled with the mass. Flaky, yellowish, or yellowish-green evacuations, containing whitish, cheesy lumps, are also met in cases of indigestion. Scanty, scybalous stools, dark-brown or black in color, and mixed with mucus, are characteristic of intestinal catarrh. Doughy, grayish, or clay-colored motions show a deficiency of bile. An intermixture of blood, altered blood-clots, and shreds of mucous membrane indicate some breach of continuity in the intestinal lining, such as occurs in follicular enteritis, typhoid fever, dysentery, and tubercular disease. Watery, almost odorless stools occur in the latter stages of entero-colitis, most offensive, carrion-like motions in both catarrhal and tuberculous ulceration of the intestines, and sour-smelling evacuations in the diarrhœa of sucklings. The discovery of worms or their ova in the stools is the certain evidence of the existence of intestinal parasites.

This outline of the changes that may take place will serve to show how much may be learned from the stools, and the importance of making a personal examination of them.

THE URINE.—It is impossible to make a definite statement as to the number of times the urine is voided by a healthy infant in each twenty-four hours. In any given case the frequency will differ very much from day to day, depending upon the temperature of the surrounding air, the amount of moisture that it contains, and so on. Sometimes it will be necessary to change the diaper every hour during the day and three or four times at night. Again, it may remain dry for six, eight, or even ten hours. Neither condition indicates disease, and between the two extremes there is a wide range of variation. Should the urine not be passed for twelve hours or more, a careful examination should

be made to discover and remedy retention. As the child grows older the frequency diminishes, and at the age of three years the number of voidings will be reduced to six or eight during the waking hours, and perhaps one at night. When the desire does arise during sleep, the child, if in a normal state, wakes up and demands the chamber, and never passes urine unconsciously. Wetting the bed, therefore, or the involuntary passage of the urine during sleep, is indicative of an abnormal condition and requires investigation. Painful micturition points to inflammation of the urethra, a narrow preputial orifice, a highly acid condition of the excretion, or stone in the bladder.

The urine of a healthy infant, while it wets, should not *stain* the diaper, the fluid being clear and almost colorless. It has a low specific gravity—1.003 to 1.006—and an acid reaction. As age advances the adult characters are more and more nearly approached, though during the whole of childhood the urine is paler and of lower specific gravity than in adult life. The normal daily amount excreted cannot be stated absolutely, but the following figures are approximate: Between two and five years, 15–25 oz.; five and nine years, 25–35 oz.; nine and fourteen years, 35–40 oz. Other characters of the urine in childhood will be considered under appropriate headings in subsequent sections.

VOMITING.—Both vomiting and regurgitation are of ready production and frequent occurrence in infancy, on account of the vertical position and cylindrical outline of the stomach at this period of life. Babies suckled at an abundant breast, and who are in perfect health, often vomit habitually. In these cases, the supply of food being large, the infant as it lies at the breast is apt to draw more than it can digest. The stomach rids itself of this over-supply by an act which more nearly resembles regurgitation than vomiting, and which must be regarded as an evidence of health rather than the reverse. There is no violent effort or retching; the material ejected is the breast-milk alone, either entirely unaltered or slightly curdled; and there are no symptoms of nausea, such as paleness, languor, and faintness. In older children vomiting may also occur after the stomach has been overladen. If the act be followed by relief from the general distress, headache, and epigastric pain, it must not be regarded as a symptom of disease.

Vomiting attended with the train of symptoms embraced under the term *nausea* is not a pathognomonic symptom. It may indicate disease of the stomach, of the intestines, of the lungs and pleura, and of the brain, or it may be a prodrome of one of the eruptive fevers. Which condition is present can only be determined by watching the case. The character of the ejecta is more definite. For instance, the expulsion of mucus is a symptom of gastric catarrh. The regurgitation of mouthfuls of curdled milk, partially digested food, and liquid so sour that it causes a grimace to pass over the face, is an indication of dyspepsia, with fermentation and the formation of acid. The appearance of lumbricoid worms in the vomit—a not infrequent occurrence—of course shows conclusively the existence of these parasites in the alimentary canal.

3. PHYSICAL EXAMINATION.

The methods of physical exploration in children are identical with those employed in adults, and the results do not differ in kind. Since, however, the object of exploration is to elicit the greatest amount of information with the least possible disturbance of the child, and as this very disturbance alters the character of some of the information obtained, it is well to adopt a somewhat different order of examination, and one which at first sight may seem irregular.

Thus it is best first to ascertain the character of the respiration and the pulse, then to strip the body to determine the degree of muscular development and the condition of the skin, next to investigate the physical condition of the lungs, heart, and abdominal organs, and last of all to examine the mouth and throat. In this order, then, the normal, as well as the more prominent abnormal, features connected with the different organs will be considered.

THE RESPIRATION.—In children the respiration is chiefly *abdominal* in type, irrespective of sex, and it is not until just before the age of puberty that the movements in the female change, becoming *superior costal*. Consequently, in estimating the number of movements per minute, it is best to place the fingers lightly on the epigastrium. The count should always be made by the watch, and the most convenient time for the observation is while the child sleeps.

Soon after birth the number of movements per minute is 44, between the ages of two months and two years, 35, and between two and twelve years, 23. During sleep the frequency is reduced about 20 per cent.

Children under two years, while awake, breathe unevenly and irregularly. In sleep there is greater regularity. After the second year the movements become steady and even. All children, however, but particularly the very young, are subject to a great increase in the rapidity of respiration under excitement, either muscular or mental.

Accelerated breathing may be caused by an elevation in the body temperature, by an interference with the blood aëration, and by thoracic or abdominal pain. As the increase in frequency may be unattended by any apparent effort or true dyspnoea, it is well to make a rule of counting the respirations in every case in which the diagnosis is doubtful.

Diminished frequency is noted in certain brain affections, as in chronic hydrocephalus, and in the later stages of tubercular meningitis. In such cases the rhythm may be greatly altered—a *tidal* form being assumed; this is termed “Cheyne-Stokes respiration.” Another form of breathing, in which the alteration is mainly in the rhythm, is termed *expiratory* respiration. It is characterized by the pause coming between inspiration and expiration, instead of between expiration and inspiration, as is the normal rule. This alteration occurs most frequently in young children, and is an evidence of dangerous pulmonary embarrassment.

Perfectly healthy children breathe through the nose, and so softly that it is difficult to hear the breezy sound of the ingoing and outgoing air. A dry, hissing sound or a moist sound of snuffling indicates partial obstruction of the nasal passages; oral respiration, complete occlusion. Difficult breathing with prolonged inspiration—*inspiratory dyspnoea*—shows an impediment to the entrance of air into the lungs and indicates laryngeal obstruction, due, most commonly, to spasm or to the formation of false membrane. In such cases the inspiratory act is also attended by a loud, piping, or rasping sound. Labored breathing with prolonged wheezing respiration—*expiratory dyspnoea*—occurs when the escape of air is impeded. The causative lesion is to be found, not in the larynx, but in the lungs. It may be a bronchial catarrh with excessive secretion, emphysema, or asthma. In both forms of dyspnoea the movements are slow as well as difficult, and a combination of the two forms is met with in cases of marked laryngeal stenosis.

Yawning, if it recur frequently, denotes great failure of the vital powers.

THE PULSE.—To obtain any reliable data from the pulse it must be felt while the patient is perfectly quiet. The best time is during sleep, but if the child cannot be caught in this condition, advantage may be taken of its placidity while nursing at the breast, feeding from a bottle, or amused by a toy.

With very young infants it is sometimes impossible to feel the beat of the radial artery, and it is necessary to ascertain the frequency of the pulse by directly auscultating the heart. After the second month palpation of the pulse at the wrist in the ordinary way presents no difficulties.

The child's pulse differs from the adult's by being much more frequent, more irregular, and more irritable, and necessarily of smaller volume.

The frequency, or the number of beats per minute, varies with the age. The following is the average rate:

From birth to the second month	160 to 130
From the 2d to the 6th month	130 to 120
" 6th " 12th "	120 to 110
" 1st " 3d year	110 to 100
" 3d " 5th "	100 to 90
" 5th " 10th "	90 to 80
" 10th " 12th "	80 to 70

These figures represent the pulse in a waking but passive state. During sleep the frequency is less. Thus, between the second and ninth years there are about sixteen beats less per minute while asleep than when awake; between the ninth and twelfth years, eight less; and between the twelfth and fifteenth years, only two less. Below the age of two years the disparity is even greater. The irregularity of the pulse in childhood is confined to an alteration of the rhythm. It is most marked in infants, and is greatest during sleep, when the pulse is slowest. The feature of irritability—that is, the facility with which its frequency is increased by muscular activity and mental excitement—is greater in proportion to the youth of the child. A rise of 20, 30, or even 40 beats a minute is not uncommon in early infancy under the excitement of the slightest effort or disturbance. On account of these wide variations in health little symptomatic meaning need be attached to alterations of the rhythm and frequency while unassociated with other abnormal features. When so associated they become important in diagnosis.

Increased frequency is a constant attendant of the febrile state. The extent of the increase corresponds with the degree of elevation of the temperature, though the pulse curve always runs higher than the temperature curve. The more frequent the pulse the higher the fever is the rule, but in estimating the prognostic value of the increase the law of the fever in question must be taken into consideration. For example, in scarlatina a pulse of 160 is usual and not indicative of special gravity, whereas in measles the same degree of acceleration would be abnormal and show great danger. Jaundice and parenchymatous nephritis are accompanied by a diminution in the rate. Irregularity is met with in diseases of the brain and heart, and sometimes in nervous and anæmic children.

The TEMPERATURE must be estimated before removing the clothing, and a clinical thermometer must always be used. The instrument is usually placed in the rectum or groin¹ of the infant and young child; in the axilla or mouth of an older and more controllable child. It should remain in position from one to five minutes, according to the delicacy of the instrument.

During the first week of life the temperature fluctuates considerably. After that the puerile norm—98.5° to 99° F.—is established, but until the fourth or fifth month it is greatly influenced by healthy causes of variation, the fluctuations ranging between 0.9° and 3.6°. By the fifth month regular morning and evening oscillations begin and certain definite laws are followed. There is a

¹ The rectal temperature is normally 1° higher than the axillary; that of the groin about 1° lower.

fall in the evening of 1° or 2° . The greatest fall occurs between 7 and 9 P. M., and the minimum is reached at or before 2 A. M. After 2 A. M. there is a gradual rise, the maximum being reached between 8 and 10 A. M. Throughout the day the oscillation is trifling. These variations are independent of eating and sleeping.

In disease there may be either a rise above or a fall below the normal standard. Fever is always associated with an elevation of the temperature. Rapid and transient rises attend slight catarrhs and passing indigestions; prolonged rises, inflammatory and essential fevers. The *degree* of elevation marks the type of the pyrexia. This is moderate when the mercury stands at 102° , severe at 104° or 105° , and very grave above 107° . The *duration* of the elevation and the peculiar *range* of the oscillations—for there are oscillations in disease as well as in health—determine the nature of the fever. The febrile oscillations differ from the healthy in that the lowest marking is noticed in the morning, the highest in the evening. *Variations* in the typical range of any given fever are important prognostic omens: a sudden fall of temperature, together with improvement in the general symptoms, indicates the beginning of convalescence; a similar fall, with an increase of the general symptoms, is a precursor of death. When the morning temperature is equal to that of the preceding evening, there is great danger; if higher, greater danger still. Marked remission in continued fevers is generally a forerunner of convalescence.

Abnormal depression of temperature is occasioned by hæmorrhage and by the loss of fluids in profuse watery diarrhœa. It is also met with in anæmia, in atrophy from insufficient nourishment, in diseases of the heart and lungs attended by imperfect blood-aëration, and it constantly attends collapse and the death agony. A maintained temperature of 97° F. is dangerous in children, and for every degree of reduction below this point the risk to life is more than proportionately increased.

THE GENERAL DEVELOPMENT.—The healthy child under two years of age is plump of body and round of limb, with well-developed fat cushions and firm flesh, and with the head and abdomen large in proportion to the rest of the frame. As age advances the figure gradually assumes the characteristics of adolescence.

To be robust, the newly-born child must have a certain average size and weight. Subsequently, under normal circumstances, there is a regular rate of increase in both of these respects. At birth the length is about 19 inches. Growth is quickest in the first weeks of life. In the first year there is an increase of from 5 to $6\frac{1}{2}$ inches; in the second, from $2\frac{2}{3}$ to $3\frac{1}{3}$ inches; in the third, from $2\frac{1}{3}$ to $2\frac{2}{3}$ inches; in the fourth, about 2 inches; and from the fifth to the sixteenth year the annual growth amounts to from $1\frac{2}{3}$ to 2 inches. The average weight at birth is from 6 to 8 pounds. The daily increase in weight should range from $\frac{1}{4}$ to $\frac{3}{4}$ of an ounce. With these data it is quite possible to estimate what should be the normal size and weight of a child at any age. Consequently, if, on being measured and weighed, he be found to fall short of the normal standard, it is proper to infer the existence of some fault in the nutritive processes—a conclusion still further borne out by a want of rotundity of outline and by flabbiness of the muscles.

The age at which the child sits erect, at which it walks, and at which the anterior fontanelle becomes ossified are points closely connected with the subject of development and nutrition. For some time after birth the child, if noticed while sitting upon the lap, will be observed to hold the head and shoulders forward or to “stoop” a little, the spine from the cervical region

to the sacrum forming a continuous curve, with the convexity directed backward. Toward the end of the eighth month the position begins to become more erect, and in a few weeks is perfectly so, the spine assuming an almost perpendicular line. Any marked delay in this change indicates general debility. At the end of the fourteenth month the child should be able to walk alone. The spine then assumes the S-like curve seen in healthy adults. A delay in walking may be due to systemic weakness or infantile paralysis affecting one or both legs. If the walking be done on the toes chiefly, if the gait be limping, and especially if knee-pain be complained of and manipulation of the limbs causes suffering, the chances are that hip-joint disease is commencing. The anterior fontanelle should be ossified or completely closed at some period between the fifteenth and twentieth months. The closure is much retarded in rickets, which is pre-eminently a disease of malnutrition. Hydrocephalus has a like effect. In a state of health the opening, while still membranous, is level with the cranial bones or very slightly depressed. Conditions of systemic exhaustion cause marked sinking, and this depression is one of the best indications of the necessity of stimulation. Bulging of the fontanelle is a symptom of chronic hydrocephalus.

CONDITIONS OF THE SKIN.—In addition to the characters already described, the skin of a healthy child has a velvety smoothness and softness, a scarcely perceptible moisture, and a great degree of elasticity.

“Mucous disease” is attended with a dry, harsh skin, which is muddy in color, and covered, especially on the extensor surfaces of the arms and legs, by a more or less thick layer of exfoliating epidermis. Chronic abdominal affections, particularly tuberculosis of the intestines and mesenteric glands, lead to harshness, acridity, scurfiness, and a wrinkled appearance of the skin covering the abdomen and thorax, with enlargement of the superficial abdominal veins. Protracted diarrhoea, and, still more, vomiting combined with diarrhoea, cause absorption of the subcutaneous fat and wasting of the muscles. The skin becomes too large for the body, is dry, harsh, discolored, and so inelastic that it falls into wrinkles over the joints when the limbs are moved, and if pinched up retains the fold for a long time. The condition of general atrophy popularly known as “marasmus” presents these features most strikingly. Dryness is a concomitant of the febrile state; excessive moisture, of prostration and collapse. Eruptions appear upon the integument in the skin diseases proper, in the exanthemata, in constitutional syphilis, and in certain digestive disorders. Œdema of the subcutaneous connective tissue may be due to affections of the heart, liver, or kidneys. The cardiac variety usually shows itself first in the feet; the renal, in the eyelids; the hepatic, in the feet and legs, secondarily to ascites.

While examining the surface it is well to look for enlargement of the superficial lymphatic glands and swelling of the joints. The former occurs in tuberculosis and syphilis; the latter, in rheumatism.

EXAMINATION OF THE ABDOMEN.—To examine this portion of the body, the child, still stripped, must be placed on its back and kept as quiet as possible. Palpation or percussion should never be made with cold hands.

The abdomen of a healthy child is prominent, uniformly soft, yielding, and painless to the touch, and to percussion gives a tympanitic sound, varying in tone according to the region percussed. The tympanitic note is lowest in pitch over the epigastric and left hypochondriac regions, the seat of the stomach; highest over the umbilical region, the position of the small intestine.

In disease *inspection* reveals any disproportion in the size or form of the abdomen, the state of the integuments, of the superficial veins, and of the

umbilicus. *Palpation* shows the temperature, pliability, moisture, and tension of the walls, and the presence or absence of tenderness, of fluctuation, and of enlargement of the mesenteric glands and other solid viscera. *Percussion* serves to demonstrate the nature of enlargements, whether due to accumulation of gas or liquid or to solid growths. By it, also, the outline and size of the liver and spleen may be determined.

Distention of the abdomen is, in the vast majority of instances, due to flatulence. In this condition the skin feels tense, the umbilicus is level or slightly prominent, there is no tenderness on pressure, and percussion is markedly tympanitic. Drum-like distention, with great tenderness, and muffled tympanitic percussion-note occur in general peritonitis. Uniform distention, again, may be due to ascites. The abdomen is barrel-shaped, painless to the touch, and there is extended fluctuation. Percussion is dull over the position of the fluid, but in nearly every instance there is an area of tympany which *changes* its position. Localized distention may be traced to gaseous accumulation, to enlargement of the liver and spleen, to fecal accumulation, to circumscribed peritonitis, and to distention of the bladder. Collections of gas are always tympanitic on percussion. The extent of liver dulness is to be estimated by percussion, or palpation with the *warmed* hand. An enlarged spleen may be felt by placing the fingers of the right hand on the back, directly below the twelfth rib and outside of the lumbar muscles, the fingers of the left on the abdomen, directly opposite, then bringing the hands toward one another. The fact that both the liver and spleen, though still unenlarged, may be more readily felt than natural when pressed downward by the diaphragm, must not be overlooked. A fecal accumulation is distinguished by the absence of tenderness, by the oblong shape of the tumor, by the situation in the region of the transverse or descending colon, to which its long axis corresponds, and by its shape being capable of some modification by pressure. Percussion over such a mass is dull. Distention of the bladder gives rise to a bulging tumor in the hypogastric region, which is elastic to the touch and dull on percussion.

A shrunken or scaphoid condition of the abdomen is met with in serious brain affections, notably tubercular meningitis, also in entero-colitis, follicular enteritis, and dysentery.

Tenderness to pressure indicates inflammatory lesion of the intestines. The presence or absence of this sign in an infant can be determined by forcing the attention, by bringing it before a strong light, for instance, and then making pressure on the abdomen. If crying be produced, there is tenderness; if not, the reverse.

EXAMINATION OF THE CHEST.—The stethoscope and pleximeter are unnecessary in examining the lungs. In the case of the heart the former may be occasionally required to localize murmurs. When used, it is better to give the instrument to the child to handle and become familiar with before application. The thoracic end must never be adjusted without being warmed. The quieter the patient, the more complete and satisfactory will be the results of the exploration. Unfortunately, though, it is too often necessary for one to do the best possible in the midst of cries and struggling. However, by skilfully seizing opportune moments much reliable information may be gained.

The steps of the examination are—first, inspection; second, auscultation; third, palpation; and fourth, percussion. The reason for making the order different from that practised in adults is to place the most disturbing element last. Mensuration and succussion are infrequently resorted to in children. If required, they are best postponed until the end of the examination.

Inspection.—The sitting posture, the child being stripped and in a good light, is the best for this process. Note is to be taken of the shape of the chest, the character of the breathing, and the position of the apex-beat of the heart.

In the new-born baby the chest is nearly circular in shape; later, the lateral diameter considerably exceeds the antero-posterior. The intercostal spaces are poorly marked, and the scapulæ lie so close that their outline is scarcely perceptible. The circular shape of the chest allows of little lateral expansion, and for this reason the respiration is chiefly abdominal in type. Together with the movement of the abdominal walls, every act of inspiration is attended by a certain amount of recession of the lower part of the chest-walls, the yielding ribs being forced inward by the pressure of the external air before they can be sufficiently supported by the expanding lung. The rise and fall of the cardiac apex can be seen—except when there is a great accumulation of fat—a short distance below and to the right of the left nipple.

Disease may alter all of these conditions. The tuberculous diathesis is characterized by a small chest, and one which has either the *alar* or the *flat* shape. In rickets the thorax becomes irregularly triangular in outline. Emphysema causes a barrel-shaped chest, with stooping shoulders and round back. Pleuritis with large effusion produces bulging of the affected side, and sometimes prominence of the intercostal spaces. After absorption has taken place there may be marked retraction, sinking of the interspaces, falling of the shoulders, and curvature of the spine toward the healthy side. Cessation of the costal respiratory movements indicates inflammation of the lung or pleura or a large pleuritic effusion; cessation of the abdominal play, inflammation of the peritoneum or of the intestines: excessive ascites and gaseous accumulations produce the same effect. Rachitic softening of the ribs, and those diseases of the lungs which offer a direct obstacle to the entrance of air, are associated with a great increase in the normal recession of the lower portion of the chest on inspiration. The position of the apex-beat is altered by cardiac diseases, by pleuritis, and occasionally by gaseous distention of the stomach. When the left ventricle is enlarged, it is shifted downward and to the left. Transmitted epigastric pulsation shows enlargement of the right ventricle. An extended impulse is not necessarily a sign of disease, since the chest-walls are so elastic in childhood that the normal impact of the apex is apt to affect a wide area. The effusion of pleurisy pushes the heart to the right or left, while the retraction, after absorption or evacuation, draws it in one or other direction. The apex is pushed upward and to the left in gastric flatulence. Emphysema, by pushing the heart away from the thoracic wall, diminishes or hides the impulse.

Auscultation.—With infants the back of the chest is most conveniently ausculted when the child is held in the nurse's left arm, with his breast against hers, his chin resting upon her left shoulder, his left arm around her neck, and his head kept in position by her disengaged hand; the front, when reclining on the back on a pillow; the sides, when sitting upright on the lap, first one arm and then the other being lifted up to allow the observer's ear to be applied. Older children may be made to take the same position as adults. It is not sufficient to auscult the posterior aspect of the thorax alone, as is stated by some authors. The whole chest should be examined, particularly in doubtful cases. The signs of croupous pneumonia are most frequently discoverable at one or other base, posteriorly; the friction-sound of pleuritis at the junction of the middle and lower third of the chest, laterally; and the signs of emphysema at

the apices, anteriorly. Therefore, unless the exploration be thorough, important lesions may be overlooked.

In healthy infants the inspiratory act in ordinary breathing is superficial, and the respiratory murmur, as a consequence, feeble. If, however, a deep inspiration be taken, a frequent occurrence under excitement and during crying, the murmur becomes loud, or *puerile*. After the age of two years puerile respiration is habitual. The breathing is loudest over the anterior, lateral, and posterior inferior regions of the thorax; faintest over the scapulæ and the præcordial area. Sometimes the expiratory element is wanting in young children over the lower posterior portions of the lungs. In the interscapular region there is often an approach to the bronchial type of breathing. If the child speaks, cries, or coughs while the ear is applied to the chest, a muffled rumbling sound, the normal vocal resonance, will be heard. At the same time vibration of the walls, the vocal fremitus, can be felt.

The cardiac sounds are readily heard when the ear is placed on the præcordia. In young infants the examination is somewhat difficult, but after the first year, the circulation becoming slower and more regular, there is little trouble in distinguishing the sounds, and even slight alterations in them. The first sound is longer and graver than the second, the rhythm is ordinarily quite regular, and the area of distribution is extended.

Palpation.—In practising palpation the palmar surface of the well-warmed hand must be applied to the naked chest. This method of exploration is useful as a means of determining the number of respiratory movements, the degree of expansion of the thoracic walls, the position of the cardiac apex-beat, the presence or absence of painful regions and of pleural or bronchial fremitus, the existence of fluctuation in the intercostal spaces, and the character of vocal fremitus.

Percussion.—In percussing the different surfaces of the chest the child must be placed in the same position as for auscultation. When contrasting the two sides, percussion should be made in identical regions and during the same period of the respiratory movement. Babies when constrained or when disturbed hold their breath in the intervals of crying, and as they always do so at the end of an inspiration, this is a favorable time to seize for the comparative examination. The percussion strokes must be lighter than in the adult, but in other respects the operation in no wise differs.

In health the resonance will be found to correspond closely with the respiratory murmur. Thus in infants under one year, the respiratory murmur being feeble, percussion is rather insonorous, but so soon as puerile respiration becomes established the resonance is uniformly intense. With the exception of this greater intensity the sound is exactly similar to that obtainable in adults. It is always attended, too, by a sensation of elasticity, appreciated by the finger used as the pleximeter.

Different portions of the thorax possess, normally, different degrees of sonorousness. In front, the right side is markedly resonant from the clavicle down to the fifth interspace or the upper border of the sixth rib in the mammary line, where the liver dulness begins. On the left side the resonance is equally intense, but it is encroached upon by the gastric tympany, which extends upward as high as the seventh or sixth rib, as well as by the area of cardiac dulness. The latter is never so decidedly marked as in adults. Laterally, both axillary regions are very resonant. The upper portions of the infra-axillary regions are a degree less resonant, and the lower portions are dull on account of the presence of the liver on the right and the spleen on the left side. The superior border of the liver dulness is found in the seventh interspace, or

at the eighth rib ; that of the spleen, at the upper edge of the ninth rib. Gastric tympany may supplant the pulmonary resonance over the left infra-axillary region. Posteriorly, there is little resonance in the scapular region, particularly the supraspinous portions. Over the interscapular space the sound improves, but it is less resonant than anteriorly or laterally. Over the infra-scapular regions the resonance is but little less pure than in front, until the tenth rib is reached on the right side and the liver dulness is again met with. On the left side the resonance extends to the very base, the posterior splenic dulness being detected with difficulty. The right base is, therefore, naturally less resonant than the left, and this difference is especially marked during expiration, the liver rising higher at that time.

Affections of the lungs produce various alterations in the percussion sound. The chief of these are the substitution of tympany, of dulness, and of flatness for the normal resonance, and of increased resistance to the finger for elasticity. Cardiac diseases cause changes in both the extent and the shape of the area of præcordial dulness.

EXAMINATION OF THE MOUTH AND FAUCES.—This portion of the examination is most apt to cause crying, but it must never be omitted. In infants gentle pressure of the fingers upon the chin is sufficient to cause wide opening of the mouth. An older child will frequently open the mouth when requested, but if he refuse, some smooth, flat instrument may be inserted in the mouth, and downward pressure made upon the tongue, when the jaws will be widely separated. The fauces can sometimes be seen by directing the mouth to be opened wide and the tongue to be alternately protruded and retracted, or a prolonged sound of “Ah” to be made. With the refractory, and always with infants, the tongue has to be held down by a spoon-handle or tongue-depressor.

The healthy oral mucous membrane has a deep pink color and is smooth, moist, and warm to the touch. The color is deeper on the lips and cheeks, lighter on the gums. The latter, up to the sixth month, as a rule, have a moderately sharp edge. Subsequently, the edge begins to broaden and soften, and the color of the investing mucous membrane deepens to a vivid red, and becomes hot as the teeth begin to force their way through. The first, or *milk teeth*—so called from their color—are twenty in number, all told, ten to each jaw ; the two lower central incisors, the first of the set, make their appearance at some time between the fourth and seventh months, the others following at stated intervals.¹ The permanent teeth, thirty-two in number, begin to appear about the sixth year.

The tongue should be freely movable. It is pink in color, and the dorsum, or upper surface, marked in the centre by a slight longitudinal depression, has a velvety appearance, and is soft, moist, and warm to the finger. The hard palate is roughened anteriorly by transverse ridges. The soft palate is smooth, and its mucous membrane is paler than that of the rest of the mouth. The fauces, on the contrary, are redder. In the triangular recess between the half-arches of the palate the tonsils can always be seen. They should be about the size and shape of almond-kernels, and they present a number of circular openings, the orifices of pouches into which the follicles open. The uvula is short and tongue-shaped. The posterior wall of the pharynx should be red, smooth, and moist.

Disease produces a great variety of changes in the mouth, tongue, and fauces. Fever makes the mouth hot and dry and causes the tongue to be frosted or coated. Affections of the gastro-intestinal tract are always attended by coating of the tongue, and the various appearances of this coating are of

¹ See article on *Dentition*.

important diagnostic and therapeutic significance. Inflammation of the mouth itself reddens the mucous membrane, makes it hot and tender to the touch, increases its moisture, alters the surface of the tongue, and leads to the formation of aphthæ, to ulceration, and even to gangrene. The eruptions of scarlet fever, measles, varicella, and varioloid make their appearance first on the mucous membrane of the palate and fauces. Finally, the conclusive evidences of diphtheria and of the various tonsillar affections are found in the fauces.

Irregular dentition indicates faulty nutrition; delayed dentition, rickets; and certain peculiarities in the formation of the permanent teeth, constitutional syphilis.

II. THE GENERAL MANAGEMENT OF CHILDREN.

1. FEEDING.

The whole question of feeding bears so close a relation to age that it is necessary to study it from the standpoint of the two stages of a child's life already mentioned.

An infant may be fed in one of three ways: 1st, from the mother's breast; 2d, from the breast of a wet-nurse; and 3d, from a bottle by the method known as artificial or hand-feeding.

1st. *Feeding from the Maternal Breast.*—This, being the natural, is the proper method of nourishing the human infant; and every mother who is able should nourish her child solely from her breast up to the age of eight months, and partially to the end of the first year, or, failing in either limit, so long as possible.

The infant should be put to the breast as soon as the mother has recovered somewhat from the fatigue of labor—some four or eight hours after birth. Of course no milk can be drawn at this early date, but the babe gets a small quantity of colostrum, which affords sufficient nourishment, and from its laxative properties clears out the infant's intestinal canal. This, too, is of great advantage to the mother, for it ensures proper uterine contraction, draws out the nipples, and encourages the formation of milk. Put the child to the breast every two hours while the mother is awake, and up to the fourth day there need be no fear of starvation. Usually on the fourth day milk is secreted and regular lactation commences. Before this time the administration of gruel or any form of artificial food is more than useless, as it lessens the activity of sucking and frequently deranges the stomach.

Many untrained mothers make a failure of nursing because they know nothing of the manner of giving suck; of the length of time the child should be kept at the breast; of the proper time for, and interval between, feedings; and of the importance of regularity.

While nursing the infant must be held partly on its side, on the right or left arm according to the gland about to be drawn, while the mother must bend her body forward, so that the nipple may fall easily into the child's mouth, and steady the breast and regulate the flow of milk with the first and second finger of the disengaged hand placed above and below the nipple. Each of the breasts should be drawn alternately, and a healthy child may be allowed to nurse until satisfied. Usually during the first six weeks the breast is required every second hour from 5 A. M. until 11 P. M., and in some cases once during the night; but this night-nursing should be given up as soon as possible, that the mother may secure essential repose. Regularity in meal hours is most important, and a little perseverance will form the habit of waking to suck the breast with almost the precision of the clock. This rule,

however, is not rigid, some infants requiring food less, others more, frequently. These exceptions can only be determined by observation of individual characteristics, and every mother must early learn to distinguish the cry of hunger from that due to the pain of indigestion, and avoid the dangerous practice of resorting to constant feeding as a means of pacifying crying.

After the sixth week the interval between nursings may be slowly increased until, by the fourth month, it reaches three hours. During this period, also, the time of lying at the breast may be gradually lengthened, for the quantity of milk secreted and the child's appetite and capacity for food are all augmented as the days pass by. At the end of the sixth month feeding every fourth hour suits some children well, but as a rule the three-hour interval must be adhered to from the fourth month to the end of lactation.

After the sixth or eighth month "mixed feeding"—breast- and bottle-feeding alternating—is advisable if the babe ceases to thrive on the breast alone. Otherwise, the maxim of not interfering with any course that is doing well is as applicable here as elsewhere, and the breast may be relied upon entirely until the time comes for weaning. Should additional nutriment be required, the food must be selected with due reference to age and prepared in the same manner as in regular hand-feeding.

The date of weaning cannot be fixed for all cases, since it depends upon the health of the mother and the development of the child. When the former continues to be robust and the child steadily grows and gains flesh, lactation can be prolonged until the tenth or twelfth month. If persevered in longer, the mother's strength usually begins to fail, her milk is lessened in quantity or becomes poor in quality, the child's nutrition suffers, and it grows pale, thin, and flabby, and may develop the disease known as rickets.

Weaning may be accomplished gradually or suddenly. In gradual weaning about four weeks are required to prepare for the absolute withdrawal of the breast. For instance, if suck be given every three hours from 5 A. M. until 11 P. M., or seven times a day, there should be, during the first week of preparation, one artificial feeding and six nursings daily; during the second, two and five, and so on until the breast is entirely withheld. Carefully prepared milk food, administered from a bottle, is the best substitute. At the age of ten months a mixture that ordinarily agrees well is—

Cream	f 3ss.
Milk	f 3iv.
Sugar of milk	3j.
Water	f 3iss.

Should fever or disordered digestion occur during the period of preparation, the number of artificial feedings must be reduced or the breast resumed until the disturbance be passed; then the course may be begun again and carried to its completion.

Sudden weaning is more difficult to accomplish, and is not advisable unless, while the breast is being presented, there is an absolute refusal to take artificial food, or unless the mother's health becomes so affected as to render any further sucking a positive peril to the child's life: attacks of erysipelas or of small-pox are instances in point.

The physician is often forced to decide upon the advisability of premature weaning. His decision must be made cautiously and after thorough investigation of two propositions—namely (*a*) the effect of further lactation upon the health of the mother; and (*b*) the requirements of the child.

(*a*) Lactation, being a physiological process, is not a drain upon the sys-

temic strength so long as the functions of nutrition are actively performed, but under other circumstances it very frequently becomes so. Premature weaning is necessary when the mother is attacked by any acute disease threatening dangerous temporary prostration, such as typhoid or typhus fever. A change must also be made if pulmonary consumption be developed, or, being already present, rapidly advances under the drain of milk-secretion. Usually, however, the general condition that leads to withdrawal of the breast is one of simple loss of strength and flesh on the part of the mother, and one which may often be overcome by attention to her health.

If the trouble be merely diminished milk-secretion, it may often be remedied by the free use of animal broths, chocolate, gruel, or milk, and sometimes the moderate employment of stimulants, in the form of ale and porter, may be necessary. Such tonics as malt extract, ferrated elixir of cinchona, bitter wine of iron, and the preparation known as "beef, wine, and iron," are useful when there is anæmia or when the general failure of strength cannot be overcome by food and attention to hygienic rules.

The ordinary local conditions indicating the necessity of premature weaning on the mother's account are fissures of the nipple and mammary abscess.

(b) On the part of the infant there are several indications for premature weaning. It must be done if the occurrence of pregnancy or the recurrence of menstruation renders the milk unwholesome; if the mother contract a dangerous contagious disease, as small-pox, scarlet fever, or erysipelas; if the mammary glands become inflamed; if the breast does not afford sufficient nourishment and artificial food be refused; and, finally, if dentition be markedly delayed and the premonitory symptoms of rickets appear.

Upon deciding to anticipate the time of weaning, the next point to consider is whether the infant shall be brought up by hand or by a wet-nurse.

2d. *Feeding by a Wet-nurse.*—The advantage of this mode of feeding is that the mother's milk is substituted by the milk of another woman; in other words, that natural feeding is continued—a matter of moment in all cases, and of inestimable importance with delicate children. The disadvantage consists in the difficulty of finding, in a woman belonging to the class from which wet-nurses come, all the moral and physical characters essential to a good substitute, and in the fact that a stranger is introduced into the household, often to deceive and annoy the family, and on the slightest provocation to leave her charge to fate or to the tender mercies of another of her kind. For these reasons it is preferable, in the majority of instances, to trust to careful bottle-feeding. Nevertheless, as some children must have human milk if their lives are to be saved, the rules for selecting a wet-nurse must be understood.

The woman chosen must be strong and robust, but rather spare than fat. Her bill of health must be perfectly free from hereditary tendency to mental or physical disease and from taint of syphilis, consumption, or scrofula. She must be cheerful, good-natured, active, careful, and temperate in habits. Her age should be between twenty and thirty years; she should understand the care of an infant and the manner of giving suck; her child ought to be nearly of the same age as the infant to be adopted, and she must be able to afford an abundant supply of good milk. The last quality can be estimated by inspecting the breasts, by examining some of the milk drawn by a pump, and by ascertaining the condition of the woman's own child. The breasts of a good nurse are not necessarily large, but are firm to the touch and pyriform in shape, with well-developed, prominent nipples, and with the skin distinctly marbled with large blue veins. The milk, which ought to flow readily on pressure or on suction, should be opaque and dull white in color, have a specific gravity of

1.031, an alkaline reaction, and show, when placed under the microscope, a number of minute, equal-sized fat-globules. Its quantity may be ascertained by weighing the child before and after sucking, the normal gain being from three to six ounces. There is, however, no better or more readily applied test of the quality of a nurse than the size, weight, and general development of her own child; and if it be weak and ill-nourished, no amount of fitness in other respects can warrant her engagement. Even when a woman is found fulfilling in her single person all the required conditions—a rare thing, indeed—it does not necessarily follow that her milk will suit the babe to be suckled. Then changes and new trials must be made until the desired end be attained.

3d. *Artificial Feeding.*—There are many women who, no matter how willing, are completely unable to suckle their babies, and a vast number in whom the secretion of milk fails after a few weeks or months of lactation. These must resort to a wet-nurse or to artificial feeding. Usually, they select the latter method.

To ensure success in hand-feeding—always a difficult task—it is important to make a detailed study of the following questions: *a*, the selection of a proper substitute for the natural food—the breast-milk; *b*, the quantity to be given; *c*, the method of preparation; *d*, the mode of administration; and, *e*, the means of preservation.

a. Healthy breast-milk must be taken as the type of infants' food, and the nearer an artificial substance can be made to approach it in chemical composition and physical properties the more perfect it is. Normal breast-milk has a specific gravity of 1.031. It is a persistently alkaline fluid, having a somewhat animal, usually disagreeable, and, very rarely, sweetish taste. It is bluish-white in color and thin and watery in consistence. It contains nitrogenous material (caseine), carbohydrates (milk-sugar and fat), salts, and water—all the elements essential to repair tissue-waste, to supply new material for growth, and to maintain body heat, or, in other words, to constitute a perfect aliment; and these, too, are so proportioned in the combination as to most easily and completely meet the demands.

In seeking a substitute for human milk one naturally turns to the domestic animals for the source of supply; cows' milk is usually selected, because, being plentiful, it is easily obtained and cheap.

Cows' milk (market milk) has a lower specific gravity than human milk—namely, 1.029; notwithstanding this, it is richer-looking—that is, whiter and more opaque; its reaction is slightly acid unless perfectly fresh from pasture-fed animals, when it may be neutral or alkaline. Its component ingredients are similar to those of human milk, but nitrogenous material exists in greater, the fat in somewhat less, and the sugar in far less proportion. The nitrogenous material also differs in quality, containing a much larger proportion of albumin coagulable by acids. This difference is readily tested by adding rennet to the two fluids. In the case of cows' milk the caseine is coagulated into large, firm masses, while with human milk a light, loose curd is formed. In the stomach the acid gastric juice has the same effect, producing in the first instance a coagulum most difficult to digest; in the other, one readily attacked and broken down by the gastro-intestinal solvents. These chemical and physical properties of cows' milk can be altered by various methods of preparation, and unless this be done there are few instances in which it will not prove a poor substitute for the natural food.

Condensed milk is frequently recommended by physicians, and largely used by the laity on their own responsibility. It keeps better than cows' milk, and is supposed to be more readily digested by young infants. The latter suppo-

sition is a mistaken one, and arises from the overlooked fact that condensed milk is always given dissolved in a large proportion of water, while cows' milk is too frequently used insufficiently diluted or otherwise improperly prepared. Condensed milk contains a large proportion of sugar, forms fat quickly, and thus makes large babies; sugar also counteracts the tendency to constipation—often a troublesome complaint in hand-feeding. These advantages are unquestioned, and, together with the ease of preparation, are those which place it so high in the esteem of monthly nurses. It is equally true, however, that as a food it contains too much cane-sugar, and not enough nutrient material to supply the wants of a growing baby. Infants fed upon it, though fat, are pale, lethargic, and flabby; although large, they are far from strong, have little power to resist diseases, often cut their teeth late, and are very liable to drift into rickets. It must be remembered also that condensed milk, when long kept or when packed in imperfect cans, not unfrequently undergoes decomposition, and thus becomes utterly unfit for use. For a temporary change of diet, however, and as a substitute during travelling or under circumstances in which sound cows' milk cannot be obtained, it may be resorted to with advantage.

The farinaceous substances so often selected, especially by the poor, to replace breast-milk, are not only bad foods, but have both directly and indirectly a deleterious effect upon the processes of nutrition. They are bad for two reasons: First, they differ materially in chemical composition from human milk. For example, in arrowroot, which is the favorite, the proportion of the tissue-building to the heat-producing element is as one to twenty, while in human milk it is about one to five. Secondly, the heat-producing principle, starch, must be converted into sugar before it can be absorbed. This change is accomplished in the body by the saliva and pancreatic juice—secretions that are not fully established until the fourth month. While the starch lies undigested in the gastro-intestinal canal it is subject to fermentation, resulting in the formation of irritant products that rapidly induce catarrh of the mucous membrane—a condition directly interfering with the digestion and absorption of food. Again, perfect nutrition demands rapid waste and removal of effete tissues as well as repair of the same. This is effected by oxidation. Now, sugars are known to have a much greater affinity for oxygen than albuminates, and when the diet consists of farinaceous material the small amount of sugar formed and absorbed appropriates oxygen that otherwise would go toward the removal of waste, and so retards the necessary changes. Farinaceous food, as such, is never permissible before the fourth month; earlier, it is only to be employed for its mechanical action as an addition to milk preparations. This will be mentioned later.

The nutrient value of the cereals and their products as they exist in so-called "infants' foods" has been imperfectly determined. They are undoubtedly useful as mechanical attenuants, but it is very certain that none of them, unless prepared with milk, can permanently meet the demands of nutrition. At the same time, it is quite probable that the soluble albuminoid substances obtained by Liebig's process have a food value of their own, making them more serviceable than the starches.

b. The quantity of food to be allowed each day varies with the appetite and age, and the question of the correct amount in a given case must be answered by observation. Nevertheless, it is well to have some guide. (See table, page 24 *et seq.*)

After the twelfth month the quantity depends upon whether additions be made to the diet or milk food be used exclusively. When the daily amount reaches three pints, the limit of the capacity of the stomach is usually attained,

and the greater demand for nutriment, as growth advances month by month, must be met by adding to the strength of the food rather than by increasing its bulk. These two factors, strength and quantity, are intimately associated throughout the whole period of infancy, and in the earlier months a mere increase in the latter is not always sufficient to maintain the balance of nutrition.

c. The object to be accomplished in the preparation of cows' milk is to make it resemble human milk as much as possible in chemical composition and physical properties. To do this it is necessary to reduce the proportion of caseine, to increase the proportion of fat and sugar, and to overcome the tendency of the caseine to coagulate into large, firm masses upon entering the stomach. Dilution with water is all that need be done to reduce the amount of caseine to the proper level; but as this diminishes the already insufficient fat and sugar, it is essential to add these materials to the mixture of milk and water. Fat is best added in the form of cream, and of the sugars either pure white loaf sugar or sugar of milk may be used. The latter is greatly preferable, as it is little apt to ferment and contains some of the salts of milk, which are of nutritive value. Firm clotting may be prevented by the addition of an alkali or a small quantity of some thickening substance. Lime-water is the alkali usually selected. It acts by partially neutralizing the acid of the gastric juice, so that the caseine is coagulated gradually and in small masses, or passes, in great part, unchanged into the intestine, to be there digested by the alkaline secretions. As it contains only half a grain of lime to the fluidounce, the desired result cannot be attained unless at least a third part of the milk mixture be lime-water. Instead of lime-water, two to four grains of bicarbonate of sodium may be added to each bottle, or, better still, from five to fifteen drops of the saccharated solution of lime.

This solution is made in the following way :

Take of—

Slaked lime	1 ounce.
Refined sugar, in powder	2 ounces.
Distilled water	1 pint.

Mix the lime and sugar by trituration in a mortar. Transfer the mixture to a bottle containing the water, and, having closed this with a cork, shake it occasionally for a few hours. Finally, separate the clear solution with a siphon and keep it in a stoppered bottle.

Thickening substances—attenuants, such as barley-water, gelatin, or one of the digestible prepared foods—act purely mechanically by getting, as it were, between the particles of caseine during coagulation, preventing their running together and forming a large, compact mass. To prepare the former, put two teaspoonfuls of washed pearl barley, with a pint of cold filtered water, into a saucepan; boil slowly down to two-thirds and strain. The liquid obtained does not possess the disadvantages of farinaceous foods generally. To be efficient, it must be used as a diluent instead of, and in the same proportion as, water. Gelatin is prepared in the following way: Put a piece of plate gelatin, an inch square, into a half-tumblerful of cold water, and let it stand for three hours; then turn the whole into a teacup; place this in a saucepan half full of water and boil until the gelatin is dissolved. When cold this forms a jelly; from one to two teaspoonfuls may be added to each bottle of milk food. When an "infants' food" is used to act mechanically, care should be taken to select one in which the starch has been converted into maltose and dextrin by the process of manufacture.

The following table and schedule will aid in the practical understanding of the method of preparing food :

Table of the Ingredients, Hours and Intervals of Feeding, and Total Quantity of Food from Birth to the End of Seventh Month.

Age.	Cream.	Whey.	Milk.	Water.	Milk-sugar.	Salt.	Hours for Feeding.	Intervals of Feeding.	Total Quantity.
During 1st week .	f3ij	f3ij	. .	f3ij	gr. xx	. .	5 A. M. to 11 P. M. Occasionally once or twice at night.	2 hours.	f3xij.
From 2d to 6th week	f3ij	. .	f3ss	f3j	gr. xx	a pinch.	5 A. M. to 11 P. M.	2 hours.	f3xvij.
From 6th week to end of 2d month	f3ss	. .	f3x	f3x	3ss	a pinch.	5 A. M. to 11 P. M.	2 hours.	f3xxx.
From 3d month to 6th month . . .	f3ss	. .	f3ij	f3jss	3j	a pinch.	5 A. M. to 10.30 P. M.	2½ hrs.	f3xxxij.
During 6th and 7th months	f3ss	. .	f3ijss	f3ij	3j	a pinch.	7 A. M. to 10 P. M.	3 hours.	f3xxxvj.

Throughout the eighth and ninth months five meals a day will be sufficient. First meal, at 7 A. M.—

Milk f3vi.
Cream f3ss.
Milk-sugar 3j.
Water f3jss.

Second meal at 10.30 A. M.—Milk, cream, and water in the same proportion; a reliable “infants’ food,” two teaspoonfuls. Third meal at 2 P. M.—same as second. Fourth meal at 6 P. M.—same as second. Fifth meal at 10 P. M.—same as first. This gives forty fluidounces of food per diem. Instead of “infants’ food,” a teaspoonful of “flour-ball” may be added. To make flour-ball, take a pound of good wheat flour—unbolted, if possible; tie it up very tightly in a strong pudding-bag; place it in a saucepan of water and boil constantly for ten hours; when cold, remove the cloth, cut away the soft, outer covering of dough that has been formed, and reduce the hard-baked interior by grating. In the yellowish-white powder obtained almost all the starch has been converted into dextrin by the process of cooking, and the proportion of the nitrogenous principle to the calorifacient is as one to five—nearly the same as human milk. Two meals of flour-ball daily—the second and fourth—are all that can be digested. To prepare these, rub one teaspoonful of the powder with a tablespoonful of milk into a smooth paste, then add a second tablespoonful of milk, constantly rubbing until a cream-like mixture is obtained. Pour this into eight ounces of hot milk, stirring well, and it is then ready for use. The other meals should be composed of milk, cream, sugar of milk, and water, as already given. Flour-ball is best suited for infants having a tendency to too frequent and liquid fecal evacuations, as it has a somewhat astringent action, and is to be avoided in cases of sluggish bowels and constipation. Under the latter conditions a more laxative food, such as oat-meal, crushed wheat, or barley, should be employed, the quantity of each being determined by the effect to be produced.

Diet from the tenth to fourteenth month—five meals daily: First meal, 7 A. M.—

Milk	f 3viiiiss.
Cream	f 3ss.
One of the Liebig foods	3ss.
(Or barley jelly)	3ij.)
Water	f 3jss.

Occasionally, about the end of the first year a child may require a more varied and substantial diet; for example: First meal, 7 A. M.—milk mixture as above. Second meal, 10.30 A. M.—a breakfast-cupful (f 3vij) of warm milk. Third meal, 2 P. M.—the yolk of an egg lightly boiled, with stale bread-crumbs. Fourth meal, 6 P. M.—same as first. Fifth meal, 10 P. M.—same as second. On alternate days the third meal may consist of a teacupful (six fluidounces) of beef tea¹ containing a few stale bread-crumbs. A further variation can be made by occasionally using mutton, chicken, or veal broth instead of beef tea.

As much more difficulty is experienced in feeding infants during the first twelve months than during the second, it would be well to pause here to consider what had best be done in case the food described should disagree.

If, after feeding, vomiting occur, with the expulsion of large, firm clots of caseine, the effect of adding lime-water or barley-water must be tried, both being added in the same quantity as the ordinary diluent—water.

Sometimes, particularly if there be diarrhœa, boiling makes the milk more tolerable; condensed milk, too, can be employed temporarily, making, for an infant of six weeks, each portion of—

Condensed milk	3j.
Cream	f 3ss.
Hot water	f 3jiiss.

Should further alteration be necessary, goats' or asses' milk may be substituted for cows' milk, the strong odor of the former and the laxative properties of the latter being removed by boiling. The milk should be used warm from the udder.

"Strippings" is another good substitute for cows' milk. It is obtained by remilking the cow after the ordinary daily supply has been drawn, and contains much cream and but little curd. One part of strippings to two of water or an equal measure of barley-water makes an easily digested mixture.

The process of predigestion or peptonization enables us to overcome many of the difficulties encountered in bottle-feeding. Pancreatin is the agent to be employed. That manufactured under the name of *extractum pancreatis* by Fairchild Brothers & Foster of New York has proved most efficient in my hands. To accomplish artificial digestion put into a clean quart bottle five grains of *extractum pancreatis*, fifteen grains of bicarbonate of sodium, and four fluid-ounces of cool filtered water; shake thoroughly together, and add a pint of fresh, cool milk. Place the bottle in water, not so hot but that the whole hand can be held in it for a minute without discomfort, and keep the bottle there for exactly thirty minutes. At the end of that time put the bottle on ice to check further digestion and to keep the milk from spoiling. The fluid obtained, while somewhat less white in color than milk, does not differ from it in taste: if, however, an acid be added, the caseine, instead of being coagu-

¹ Beef tea for an infant is made in the following way: Half a pound of fresh rump-steak, free from fat, is cut into small pieces and put, with one pint of cold water, into a covered tin saucepan. This must stand by the side of the fire for four hours, then be allowed to simmer gently (never boil) for two hours, and, finally, be thoroughly skimmed to remove all grease.

lated into large, firm curds, takes the form of minute soft flakes or readily broken-down, feathery masses of small size. When the process is carried just to the point described, the caseine is only partly converted into peptone, but every succeeding moment of continued warmth lessens the amount of caseine until peptonization is complete. Then the liquid is grayish-yellow in color, has a distinctly bitter taste, and shows no coagulation whatever on the addition of an acid.

"Peptogenic milk powder," prepared by the same chemists, has given me even better results than the pancreatin and soda. This powder contains a digestive ferment, pancreatin; an alkali, bicarbonate of sodium; and a due proportion of milk-sugar. The mode of employment is as follows:

Take of—

Milk	f 3ij.
Water	f 3ij.
Cream	f 3ss.
Peptogenic milk powder	3j. ¹

This mixture is to be heated slowly to boiling, ten minutes being occupied, and then quickly cooled. When properly prepared the resultant, so-called "humanized milk," presents the albuminoids in a minutely coagulable and digestible form; has an alkaline reaction; contains the proper porportion of salts, milk-sugar, and fat; is not bitter in taste, being but partially peptonized, and in appearance as well as chemical composition resembles human milk.

The great advantages of partial peptonization are that the necessity for lime-water, barley-water, and thickening substances to keep apart the curd is done away with, and that, when the digestive disturbance requiring a careful preparation of food is removed, an ordinary milk diet can be gradually resumed by regularly diminishing the time artificial digestion is allowed to progress. This changes the caseine in a less and less degree, until, finally, it is taken in its natural form.

"Sterilization" is another process of importance. As milk exists in the healthy cow's udder it is aseptic—*i. e.* free from any poisonous or dangerous ingredient—but during milking and subsequent handling and transportation various foreign materials get into it and are apt to set up some injurious change. To deprive these accidentally introduced organic impurities of their activity—or, in other words, to *sterilize*—it is necessary to subject the fluid to high heat under pressure.

Several admirable implements have been devised for conducting the process; one of the most simple, made after a design of my own, is shown in Fig. 1.

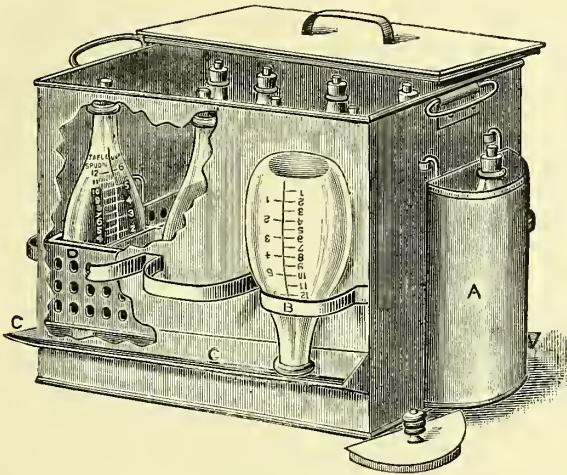
This apparatus is made of tin, and consists of an oblong case provided with a well-fitting cover, and having a movable perforated false bottom (D), which stands a short distance above the true one and has attached a framework capable of holding ten six-ounce nursing-bottles. On the outside of the case is a row of supports (B) for holding inverted bottles while drying, and at the proper distance below these a gradually inclining gutter (C) for carrying off the drip. A movable water-bath (A) is hung to the side; in this each bottle of food may be warmed at the time of administration. Ten graduated nursing-bottles are used, so that the whole supply of milk intended for a day's consumption can be prepared at once. Each bottle is provided with a perforated rubber cork, which in turn is closed by a well-fitting glass stopper.

Sterilization should be performed in the morning as soon as possible after

¹ Measure provided with jar only to be used when preparing, at once, the whole quantity of food to be given in a day.

the milk has been served. The process is as follows: First, see that the ten bottles are perfectly clean and dry; pour into each six fluidounces of milk; insert the perforated rubber corks, without the glass stoppers, however; remove the false bottom and place the bottles in the frame; pour into the

FIG. 1.



Author's Sterilizer.

case enough water to fill it to the height of about two inches; replace the false bottom carrying the bottles; adjust lid and put the whole on the kitchen range. Allow the water to boil, and, by occasionally removing the lid, ascertain that the expansion that immediately precedes boiling has taken place in the milk; then press the glass stoppers into the perforated corks, and thus hermetically close each bottle. After this keep the apparatus on the fire and the water boiling for twenty minutes. Finally, remove the false bottom with the bottles; pour out the water, replace and carry the whole, covered with the lid, to the nursery.

Milk sterilized by this process will remain sound for many days: it is especially useful in travelling, when fresh milk cannot be obtained; for use in cities during the heat of summer, when milk is most apt to undergo injurious changes; for a temporary change of food in delicate children or for those suffering from disease of the stomach or intestinal canal. It must be remembered, however, that the prolonged heating produces certain changes in the composition of the milk which make it more difficult to digest, and that on this account many children do not thrive upon it.

Another process of sterilization, suggested by Leeds, is free from this disadvantage, and has proved most useful in my practice. It consists in heating the milk, rendered feebly alkaline with lime-water or sodium bicarbonate, to 155° F. for six minutes, or, better still, of applying the same amount of heat to milk with pancreatin and bicarbonate of sodium or with peptogenic milk powder. By the latter method the milk is both predigested and sterilized; if not used at once, it must be momentarily heated to the boiling-point to check peptonization before the development of a bitter taste.

According to Rowland G. Freeman, the problem that presents itself in the sterilization of milk for food is to devise a method which shall destroy by efficient means the contained germs, and yet in the least possible degree interfere with its nutritive qualities. The experiments of Leeds show that

sterilization at the boiling-point of water causes the following modifications: the starch-liquefying ferment is destroyed and coagulated; caseine is rendered less coagulable by rennet, and is acted on slowly and imperfectly by pepsin and pancreatin; proteid matters attach themselves to fat-globules, and probably bring about a less perfect assimilation of fat; while milk-sugar, by prolonged heating, is completely destroyed. Koplik states that "from the temperature of 75° C. upward there is a separation of the serum-albumin of the milk; the caseine loses its coagulability to rennet, and at 85° C. amounts of rennet which for the raw condition of milk are found sufficient to act cease to be effective." Hueppe considers that from a physiological standpoint milk is best sterilized under a temperature of 75° C., while other experimenters have shown that temperatures lower than 100° C., if continued for a short time, will destroy a very large proportion of the germs, and will destroy with certainty many pathogenic germs which find their way into milk either from the cow or as external contaminations.

Dr. Freeman, therefore, feels satisfied that Pasteurization offers the most rational solution of the question under consideration. The elaborate and recent experiments of Yersin, Granchier, Lidoux-Libard, and Bitter show that the bacillus tuberculosis in milk will be destroyed in ten minutes by an exposure to 75° C., in fifteen minutes to 70°, and in thirty minutes to 68°. Concerning other bacteria, Van Geuns found that a few seconds' exposure to 60° would kill the cholera spirilla, the Finkler-Prior bacillus, the typhoid bacillus, and the pneumococcus of Friedländer.

It may, therefore, be concluded that a temperature of not less than 158° F. will render milk sufficiently germ-free for infant food, and that a temperature of less than 176° F. will not injure milk materially. Methods of Pasteurizing milk in bulk have been brought forward both in Germany and in this country; and now the procedure has been brought down to an easily-managed system for household use. This depends upon the theory that the temperature of the milk to be treated may be raised to about the desired point (167° F.) by immersing a certain definite quantity of milk in a properly proportioned bulk of boiling water, the source of heat having been removed. The apparatus consists of two parts, a graduated pail for the water and a receptacle for the bottles of milk. This receptacle consists of a series of seven or ten hollow zinc cylinders fastened together, which fits into the pail containing the boiling water. Each of these cylinders is large enough to hold one of the bottles of milk, the series of seven cylinders accommodating seven eight-ounce bottles, and the series of ten cylinders being intended for ten six-ounce bottles. When the bottles are in place water is poured around them to secure perfect conduction of the heat. After the water in the pail is thoroughly boiling, it is removed from the stove and placed on a non-conducting surface. The cylinders are now introduced, and the pail covered and left standing for thirty minutes, after which the milk is rapidly cooled in a refrigerator or by cold water or ice and water. Milk thus treated and put immediately into a refrigerator usually shows no change for several days.

Sometimes milk, in every form and however carefully prepared, ferments soon after being swallowed and excites vomiting, or causes great flatulence and discomfort, while it affords little nourishment. With these cases the best plan is to withhold milk entirely for a time and try some other form of food. The following are good substitutes:

Veal broth ($\frac{1}{2}$ lb. of meat to the pint)	f 3iss.
Barley-water	f 3iss.

Or, Whey f̄3iss.
 Barley-water f̄3iss.
 Milk-sugar 5ss.

For one portion; to be given every two hours at the age of two months.

A teaspoonful of the juice of raw beef every two hours will usually be retained when everything else is rejected. Such foods are only to be used temporarily until the tendency to fermentation within the alimentary canal ceases; then milk may be gradually and cautiously resumed.

When infants approaching the end of the first year become affected with indigestion, it is often sufficient to reduce the strength and quantity of the food to a point compatible with digestive powers. For instance, at eight months the food may be reduced to that proper for a healthy child of six months or even less. Here, too, predigestion of the food is very serviceable. If a few grains of *extractum pancreatis* be added to a gobletful of thick, well-boiled starch gruel at a temperature of 100° F., the gelatinous mucilage quickly grows thinner, and soon is transformed into a fluid, the starch having been rendered soluble by the action of the *pancreatin*; by still longer contact the hydrated starch is converted into dextrin and sugar. Advantage may be taken of this property to render the foods containing starch assimilable. Thus, to a mixture of barley jelly and milk—*e. g.*

Barley jelly 3ij,
 Milk sugar 3j,
 Warm milk f̄3viij,

add three grains of *extractum pancreatis* and five grains of bicarbonate of sodium, and keep warm for half an hour before administering.

The same process may be employed with food containing oatmeal, arrow-root, or wheaten flour, or in the case of meat broths, with a view of converting the starchy and albuminoid ingredients into digestible elements without materially altering the taste.

Returning to the regimen of the healthy infant, it will be found that after the fourteenth month far less change is required in the food.

Diet from the fourteenth to the eighteenth month, five meals per day: First meal, 7 A. M.—a slice of stale bread, broken and soaked in a breakfast-cup (eight fluidounces) of new milk. Second meal, 10 A. M.—a teacup of milk (six fluidounces), with a soda biscuit or thin slice of buttered bread. Third meal, 2 P. M.—a teacup of meat broth (six fluidounces), with a slice of bread; one good tablespoonful of rice-and-milk pudding. Fourth meal, 6 P. M.—same as first. Fifth meal, 10 P. M.—one tablespoonful of Mellin's Food, with a breakfast-cupful of milk.

To alternate with this: First meal, 7 A. M.—the yolk of an egg lightly boiled, with bread-crumbs; a teacupful of new milk. Second meal, 10 A. M.—a teacupful of milk, with a thin slice of buttered bread. Third meal, 2 P. M.—a mashed baked potato, moistened with four tablespoonfuls of beef tea; two good tablespoonfuls of junket. Fourth meal, 6 P. M.—a breakfast-cupful of milk, with a slice of bread broken up and soaked in it. Fifth meal, 10 P. M.—same as second.

The fifth meal is often unnecessary, and sleep should never be disturbed for it; at the same time, should the child awake an hour or more before the first meal, he must break his fast upon a cup of warm milk, and not be allowed to go hungry until the set breakfast hour.

Diet from eighteen months to the end of two and a half years, four meals

a day: First meal, 7 A. M.—a breakfast-cupful of new milk; the yolk of an egg lightly boiled; two thin slices of bread and butter. Second meal, 11 A. M.—a teacupful of milk, with a soda biscuit. Third meal, 2 P. M.—a breakfast-cupful of beef tea, mutton or chicken broth; a thin slice of stale bread; a saucer of rice-and-milk pudding. Fourth meal, 6.30 P. M.—a breakfast-cupful of milk, with bread and butter.

On alternate days: First meal, 7 A. M.—two tablespoonfuls of thoroughly cooked oatmeal or wheaten grits, with sugar and cream; a teacupful of new milk. Second meal, 11 A. M.—a teacupful of milk, with a slice of bread and butter. Third meal, 2 P. M.—one tablespoonful of underdone mutton pounded to a paste; bread and butter, or mashed baked potato moistened with good plain dish gravy; a saucer of junket. Fourth meal, 6.30 P. M.—a breakfast-cupful of milk, a slice of soft milk toast or a slice or two of bread and butter.

When sickness supervenes, all that is ordinarily necessary is a reduction of the diet to plain milk or some easily digestible milk mixture.

An important point, often neglected, is the matter of drink. Even the youngest infant requires water several times daily, and the demand increases with age. The water must be as pure as possible, and should not be too cold. In the heat of summer, however, bits of ice and water moderately cooled by ice can be allowed without harm.

The foregoing schedule must, of course, be regarded only *as an average*. Many children can bear nothing but milk food up to the age of two or even three years, and, provided enough be taken, no fear for their nutrition need be entertained. If a child be thriving on milk, he is never to be forced to take additional food merely because a certain age has been reached; let the healthy appetite be the guide.

d. Success in hand-feeding depends quite as much on the administration as upon the preparation of the food.

From birth up to such time as broth, bread, and eggs are added to the diet all the food should be taken from a bottle. Even after this, as the bottle is a comfort and ensures slow feeding, it may be allowed for milk preparations until the child, of his own accord, tires of it. The only feeding apparatus to be admitted to the nursery is the simple bottle and tip. The bottle made after my suggestion, and known as the "graduated nursing-bottle," has an interior surface free from angles, so that it is readily kept clean, and is provided with a scale for the measurement of ounces and half-ounces. It is made of transparent flint glass, so that the slightest foulness can be detected at a glance, and may vary in capacity from six to twelve fluidounces according to the age of the child. Two should be on hand at a time, to be used alternately. Immediately after a meal the bottle must be thoroughly washed out with scalding water, filled with a solution of bicarbonate or salicylate of sodium—one teaspoonful of either to a pint of water—and thus allowed to stand until next required; then, the soda solution being emptied, it must be thoroughly rinsed with cold water before receiving the food. The tips or nipples, of which there should also be two, must be composed of soft, flexible India-rubber, and a conical shape is to be preferred, as being more readily everted and cleaned; the opening at the point must be free, but not large enough to permit the milk to flow in a stream without suction. At the end of each feeding the nipple must be removed at once from the bottle, cleansed externally by rubbing with a stiff brush wet with cold water, everted and treated in the same way, and then placed in cold water and allowed to stand in a cool place until again wanted.

Next to cleanliness of the feeding apparatus it is important to insist upon the separate preparation of each meal immediately before it is to be given. The

practice of making, in the morning, the whole day's supply of food, though it saves trouble, is a most dangerous one. Changes almost invariably take place in the mixture, and by the close of the day it becomes unfit for consumption.

The food must be administered at a temperature of about 95° F. It may be heated by steeping the bottle containing the food in hot water or by placing it in a water-bath over an alcohol lamp or gas-jet.

When feeding, the child must occupy a half-reclining position in the nurse's lap. The bottle should be held by the nurse, at first horizontally, but gradually more and more tilted up as it is emptied, the object being to keep the neck always full and prevent the drawing in and swallowing of air. Ample time—say five, ten, or fifteen minutes, according to the quantity of food—should be allowed for the meal. It is best to withdraw the bottle occasionally for a brief rest, and after the meal is over sucking from the empty bottle must not be allowed, even for a moment.

e. For children residing in cities an honest dairyman must be found who will serve sound milk and cream from country cows once every day in winter, and twice during the day in the heat of summer. The milk of ordinary stock cows is more suitable than that from Alderney or Durham breed, as the latter is too rich, and therefore more difficult to digest. The mixed milk of a good herd is to be preferred to that from a single animal: it is less likely to be affected by peculiarities of feeding, and less liable to variation from alterations in health or different stages of lactation.

The care of the herd and of the milk is of great consequence. The cows should be healthy, and the milk of any animal that seems indisposed should be excluded. The cows must not be fed upon swill or the refuse of breweries, glucose-factories, or any other fermented food. They must not be allowed to drink stagnant water, and must not be heated or worried before being milked. The pasture must be free from noxious weeds, and the barn and yard must be kept clean. The udder should be washed, if dirty, before the milking. The milk must be at once thoroughly cooled. This is best accomplished by placing the can in a tank of cold spring-water or in ice-water, the water being the same depth as the milk in the can. It is well to keep the water in the tank flowing; indeed, this is necessary unless ice-water be used. The can should remain uncovered during the cooling, and the milk should be gently stirred. The temperature should be reduced to 60° F. within an hour, and the can must remain in the cold water until the time for delivering. In summer, when ready for delivery, the top should be placed in position and a cloth wet in cold water spread over the can, or refrigerator cans may be used. At no season should the milk be frozen, and at the same time no buyer should receive milk having a temperature over 65° F.

For transportation from the dairy it is safer for the family to provide two sets of small cans—one set to be thoroughly cleansed and aired, while the other is taken away by the milkman to bring back the next supply. So soon as this arrives in the morning, or in the morning and evening in hot weather, the milk should be emptied into separate and absolutely clean earthenware or glass pitchers, and these put at once into a refrigerator reserved exclusively for them. This may stand in some convenient spot near the nursery, but not in it, and especially not in an adjoining bath-room. With a good refrigerator there is no difficulty in keeping milk perfectly sweet for twenty-four hours in winter and for twelve hours in summer, except on intensely hot days; then it may be necessary to scald, slightly boil, or sterilize the whole of the supply when received, in order to prevent change.

CHILDHOOD.—Children who have cut their milk teeth may be fed for 2

twelvemonth—namely, up to the age of three and a half years—in the following way: First meal, 7 A. M.—one or two tumblerfuls of milk, a saucer of thoroughly cooked oatmeal or wheaten grits, and a slice of bread and butter. Second meal, 11 A. M. (if hungry)—a tumblerful of milk or a teacupful of beef tea with a biscuit. Third meal, 2 P. M.—a slice of underdone roast beef or mutton or a bit of roast chicken or turkey, minced as fine as possible; a baked potato thoroughly mashed with a fork and moistened with gravy; a slice of bread and butter; a saucer of junket or rice-and-milk pudding. Fourth meal, 7 P. M.—a tumblerful of milk and one or two slices of well-moistened milk toast.

From three and a half years up the child must take his meals at the table with his parents, or with some reliable attendant who will see that he eats leisurely. The diet, while plain, must be varied. The following list will give an idea of the food to be selected:

BREAKFAST.

<i>Every Day.</i>	<i>One Dish only Each Day.</i>	
Milk.	Fresh fish.	Eggs, plain omelette.
Porridge and cream.	Eggs, lightly boiled.	Chicken hash.
Bread and butter.	“ poached.	Stewed kidney.
	“ scrambled.	“ liver.

Sound fruits may be allowed before and after the meal, according to taste, as oranges, grapes (seeds not to be swallowed), peaches, thoroughly ripe pears, and cantaloupes.

DINNER.

<i>Every Day.</i>	<i>Two Dishes Each Day.</i>	
Clear soup.	Potatoes, baked.	Hominy.
Meat, roasted or broiled, and cut into small pieces.	“ mashed.	Macaroni, plain.
Bread and butter.	Spinach.	Peas.
	Stewed celery.	String-beans, young.
	Cauliflower.	Green corn, grated.

Junket, rice-and-milk, or other light pudding, and occasionally ice cream, may be allowed for dessert.

SUPPER.

<i>Every Day.</i>
Milk.
Milk toast or bread and butter.
Stewed fruit.

Fried food, highly seasoned or made-up dishes are to be excluded, and no condiment but salt is to be used. Eating, however little, between meals must be absolutely avoided. Keep a young child from knowing the taste of cakes or bonbons, or, having learned it, let him feel that they are as unattainable as the thousand other things beyond his reach, and he soon ceases to ask for them. Even a piece of bread between meals should be forbidden. His appetite then remains natural, and he will eat proper food at his regular meal hours. As to the quantity, a healthy child may be permitted to satisfy his appetite at each meal, under the one condition that he eats slowly and masticates thoroughly. Filtered or spring water should be the only drink, tea, coffee, wine, or beer being entirely forbidden.

In case of illness the diet must be reduced in quantity and quality, according to the rules that are applicable to adults.

2. BATHING.

During the first two and a half years of life a child ought to be bathed once every day. The bath should be given at a regular time, and it is best to

select some hour in the early morning, midway between two meals—ten o'clock, for instance. The tub should be placed near the fire or in a warm room in winter, and away from currents of air in summer. It should contain enough water to cover the child up to the neck when in a reclining posture, and the temperature must be about 95° F. Upon undressing the child the first step is to wet his head; then he is to be plunged into the water and thoroughly washed with a soft rag or sponge and pure, unscented castile soap. After remaining in the water from three to five minutes the surface must be well dried and rubbed with a flannel cloth or soft towel; then the body must be enveloped in a light blanket and the infant either returned to his crib to sleep or kept in the lap for ten or fifteen minutes until thoroughly warm and rested, and finally dressed. If there be repugnance to the bath, the tub may be covered over with a blanket, and the child, being placed upon it, may be slowly lowered into the water without seeing anything to excite his fears. In very hot weather, in addition to the morning full bath, the body may be sponged twice daily with water at a temperature of 90° F.; this, contrary to what might be expected, has a greater and more permanent cooling effect than bathing with cold water.

After the third year three baths a week are quite sufficient. An evening hour is now to be preferred, but the water must still be heated to 90°. About the tenth year cooler baths can be begun, from 72° to 75° being the proper temperature. The cold sponge or cold plunge is not admissible as a daily routine until youth is well advanced.

The hot bath—95° to 100° F.—is employed for various purposes, notably for a derivative action, to cause diaphoresis, to relieve nervous irritability, and to promote sleep. Whether a full bath or merely a foot-bath be required, five minutes is a sufficient time for immersion; then, with or without drying, according to the degree of sweating desirable, the whole body, or only the feet and legs in case of a foot-bath, must be enveloped in a blanket, and the child put to bed. To render these baths more stimulating, from a teaspoonful to a tablespoonful of mustard flour may be added, and the child held in the water until the *arms* of the nurse begin to tingle. It is important not to continue a hot bath too long, lest the primary stimulating effect be followed by depression.

Cold baths, by shocking the system, first produce depression; but this is temporary and is followed by reaction, during which the skin grows red and the pulse becomes fuller and stronger. They have, therefore, a general stimulant and tonic action, promoting nutrition and giving tone to the body. On account of the shock, the extent of which depends directly upon the coldness of the water, these baths must be used with caution, and are not to be employed in very young or feeble subjects. When giving a cold bath, the child must be stripped in a warm room, and thoroughly rubbed with the palm of the hand until the whole body, especially the spinal region, is reddened; he must then stand in a tub containing enough hot water to cover the feet, and be rapidly sponged with the cold water. The temperature of the latter must never be below 60°, and the addition of half an ounce of sea-salt or a tablespoonful of concentrated sea-water to the gallon renders it more stimulating and ensures a complete reaction. After the sponging the surface must be thoroughly and quickly dried with a soft towel and shampooed with the open hand until aglow.

The cooled bath may be employed with advantage in extreme conditions of hyperpyrexia. The child is first immersed in water at 95°, and this is gradually lowered to 70° by the addition of cold water, the process occupying from fifteen to thirty minutes.

3. CLOTHING.

Infants and young children have little power of resisting cold, and on this account require warm clothing. The condemnation of the fashion of allowing children to go, even while in the house, with bare legs and knees must be absolute. Occasionally during the most oppressive heat of a summer midday the legs may be left uncovered; but with this exception the rule is to keep the whole body encased in woollen underclothing. The thickness of this must vary, of course, with the season. Providing this be done, the outer clothing may be left to the taste of the mother; but all garments should fit loosely, that the functions of the different viscera may not be impeded by pressure.

The best pattern of a winter night-dress is a long, plain slip, with a drawing-string at the bottom, to prevent exposure of the feet and limbs should the child kick off the bed-covering. This should be made of flannel or the more easily washed canton flannel. In summer a loose muslin one may be put on, without the drawing-string. A flannel under-vest should always be worn at night, light gauze in summer and heavier wool in winter; care must be taken, however, to have one for night alone, discarding that worn in the daytime.

In infants under a year old a broad flannel abdominal bandage, extending from the hips well up to the thorax, or, better still, a knitted worsted band shaped to fit the form, is very useful in keeping the abdominal organs warm, aiding digestion and preventing pain.

All clothing should be changed sufficiently frequently to ensure cleanliness.

Shoes must be large, well shaped, and made of soft leather with pliable soles, so as to allow the feet to grow freely.

When dressing a child for exercise in the open air in cold weather, the outer clothing must not be put on until just before leaving the house, and removed immediately on return. It is important to protect the head from cold in winter by a close-fitting, thick cap, and from the direct rays of the sun in summer by a broad-brimmed, light straw hat. Rubber shoes are necessary in wet weather to keep the feet warm and dry while walking out of doors.

4. SLEEP.

For some time after birth infants spend the intervals between being fed, washed, and dressed, in sleep, and thus pass fully eighteen out of the twenty-four hours. As age advances the amount of sleep required becomes less, until at two years thirteen hours, and at three years eleven hours, are enough. This matter, though, is perhaps more a question of training than any other item of nursery regimen, and one cannot too soon begin to form the good habit of regularity in sleeping hours. So far as circumstances will admit, the following rules may be enforced:

From birth to the end of the sixth or eighth month the infant must sleep from 11 P. M. to 5 A. M., and as many hours during the day as nature demands and the exigencies of feeding, washing, and dressing will permit. From eight months to the end of two and a half years a morning nap should be taken from 12 M. to 1.30 or 2 P. M., the child being undressed and put to bed. The night's rest must begin at 7 P. M. If a late meal be required, the child can be taken up at about ten o'clock; but if past the age for this, he may sleep undisturbed until he wakes of his own accord some time between 6 and 8 A. M. From two and a half to four years, an hour's sleep may or may not be taken in the morning, according to the disposition of the subject; but in every case the bed must be occupied from 7.30 P. M. to 6 or 7 o'clock on the following morning. After the fourth year few children will sleep in the daytime; they are ready for

bed by 8 P. M., and should be allowed to sleep for ten hours or more. A later retiring hour than 9 P. M. ought not to be encouraged until after the twelfth or fifteenth year.

When feasible, different rooms should be used for the day nursery and the sleeping apartment. If an apartment has to be occupied during both the day and night, it must be vacated for half an hour or more in the evening and well aired before the child is put to bed. The temperature of the room must be as uniform as possible, the proper degree of heat being from 64° to 68° F.

5. EXERCISE.

A certain amount of muscular exercise is necessary for development and for the proper performance of the digestive functions. Infants before they are able to stand will use their muscles sufficiently if, when loosely clad, they are placed upon their backs in a bed and allowed to kick and turn about at pleasure. After the age of nine or ten months a healthy child will begin to creep; at the end of a year he will make efforts at standing, and from four to eight months later will be able to walk by himself; children, however, present great differences in this respect, and a delay of a few months must not be considered as abnormal. So soon as efforts at creeping are made there need be no fear that insufficient exercise will be taken; the care should be rather to prevent over-fatigue. Fresh air and sunlight are as necessary as muscular exercise. The child must be taken out of doors every day, weather permitting, after arriving at the proper age: this is four months for children born in the early fall and winter, and one month for those born in summer. In cool weather babies who are unable to walk should be taken out in a coach or in the nurse's arms for an hour in the morning and half an hour in the afternoon, while the sun is shining. In summer they may pass the greater part of the waking hours in the open air, provided they be well protected from the direct rays of the sun. Children old enough to walk may spend a longer time in the air in winter, and may be out all day in summer. But until the fourth year it is better to let them play about at will than take a long set walk. Until well advanced in childhood the house is the safest place in damp and rainy weather, when there is a strong east or north wind blowing, and when the thermometer stands below 15° F.

III. GENERAL REMARKS ON TREATMENT.

It is difficult to formulate a precise, reliable, or handy posological table; in fact, the whole matter of dosage for children is one of experience, and with practice every one makes his own dose-list in his mind, and the proper amount of a given drug for a given age requires as little effort of memory as in the case of adults. Nevertheless, as a guide to the student, Cowling's rule is serviceable—namely, the proportionate dose for any age under adult life is represented by the number of the following birthday divided by 24—*i. e.* for one year, $\frac{2}{24} = \frac{1}{12}$; for two years, $\frac{3}{24} = \frac{1}{8}$; and so on.

All powerful drugs must be given with caution to children, but opium requires the greatest care. Infants bear it only in infinitesimal proportions, and in these its use is to be avoided as much as possible; still, combined with castor oil, it is a useful drug in bad cases of flatulent colic, the average commencing dose in the first six weeks of life being not more than $\mathfrak{M}_{\frac{1}{32}}$ of the tincture (laudanum). After the second or third month the extreme susceptibility to the drug disappears, and $\mathfrak{M}_{\frac{1}{4}}$ of laudanum may be given for a dose.

Bromide of potassium, a most valuable remedy in many diseases, must be given to infants with watchfulness, as it sometimes, even in small doses, produces severe local inflammations of the skin and localized patches of soft, warty growths.

Belladonna and arsenic are illustrations of an opposite tendency, for children are very tolerant of these drugs, particularly the first. A child of four or five years can readily take from two to five minims of tincture of belladonna, and in cases in which it is necessary to administer arsenic to choreic children of six years and upward a commencing dose of five minims of Fowler's solution may often be given three times daily, and a considerable increase in this be attained if required. Such initial doses are, however, occasionally productive of the symptoms of mild arsenical poisoning, and therefore it is well to begin with one- or two-minim doses and increase rapidly. This rule applies especially to children belonging to the wealthier classes, for these, like their parents, are much more sensitive to drugs than hospital patients—an undoubted physiological fact of wide bearing.

Alcohol is frequently indicated and is of great value, but it must be used with judgment. It is most useful in broncho-pneumonia, severe febrile conditions; in the prostration following measles, diphtheria, and whooping cough; and in the collapse that frequently attends severe thoracic or abdominal disease.

All drugs should be made as palatable as possible.

In conclusion, it must be remembered that children do not often require energetic treatment with drugs. Proper feeding and hygiene are of most importance in the management of disease in early life.

Antipyretics.—Antipyrine especially, and phenacetin to a less degree only, must be used with extreme caution in the febrile affections of early life, on account of their marked tendency to produce cardiac depression. Sponging the surface at proper intervals with tepid or cool water is a much safer method of reducing temperature, but in every instance the *law of the temperature-curve of the disease under treatment* must be taken into consideration; and it is a safe rule not to interfere unless the temperature excess be great and maintained. For example, in pneumonia, a disease in which antipyretic drugs are especially dangerous and most frequently abused, an evening temperature of 105° is to be expected, and unless maintained is neither cause for alarm nor for the use of a powerful drug that tends to sap the strength of the cardiac muscle, the very keystone of the bridge leading to recovery.

THE CHEMISTRY OF MILK AND OF ARTIFICIAL FOODS FOR CHILDREN.

BY ALBERT R. LEEDS, PH. D.,

HOBOKEN.

I. THE CHEMISTRY OF MILK.

THE peculiar adaptation of milk to the feeding of the young depends upon its unique combination of chemical and physical properties. It contains in well-balanced proportions the three essential elements of nutrition—the nitrogenous, or tissue-building; the carbohydrate, or heat-giving; and the fats. Along with these are a sufficiency of saline substances to carry on the chemical metamorphoses of cell-formation, of secretion and excretion, and an ample supply of water as the universal solvent. These substances are held partly in a state of solution, partly in a state of semi-solution, conferring upon milk its slightly colloidal consistency, and partly in suspension, producing its appearance of density and opacity. But it contains no waste material like the indigestible fibre and cellulose of flesh, fruit, and vegetables. Neither does it exhibit a development of one or two elements of nutrition at the expense of the third, as is the case with all other foods,—even eggs, which most nearly approach milk in this respect, not being excepted. Finally, almost no preparation before, during, or after swallowing is requisite for the absorption of milk through the rudimentary digestive apparatus of the young.

The chemistry of woman's milk can be well and effectively studied for our present purposes only in connection with that of cow's milk. For at the very outset a peculiar difficulty is experienced in attempting to procure a sample of the former, which does not exist in the case of the latter. Some sort of a breast-pump or similar appliance must be used, and this unnatural process yields at the best but a partial sample. This fact explains many of the great and anomalous variations exhibited in the analyses. It also renders the conclusions drawn from an isolated analysis of little value; and in practice it is wiser to base any conclusions as to the sufficiency and quality of the breast-milk upon the condition and yield of the gland, upon the physical condition and nutrition of the mother, and, most of all, upon the development of the child and its deportment in nursing.

On the other hand, innumerable analyses of complete samples of cow's milk exist, embracing every variety of breed, under every condition of climate, age, culture, and feeding.

Cow's Milk.—On no other article of food has such elaborate care been expended, both as to its production and chemical investigation. Most civilized communities have enacted laws to protect its purity, and recognize no evidence in courts of law except when substantiated by adequate chemical testimony.

Similar investigations are being constantly made with a view of so adjusting the feeding and the breed as to obtain the largest quantity of milk or the greatest richness, or both. Beginning with cattle of small, imperfectly-developed udders, the cow has become through generations of culture the incomparable milk-secreting animal of modern nations, and has so far displaced the ass, goat, mare, and others that it is useless to consider their milk as an available substitute.

For similar reasons, the cow's milk which must be considered from the standpoint of general dietetics is such sound, whole country milk as is ordinarily supplied by reputable dealers. It is useless to quote the analyses of the milk of Alderney, Jersey, and Guernsey cattle, obtainable only by the few; and when obtained, such milk, with its higher percentage of proteids and its greater liability to variation from idiosyncrasy in condition or health of individual cattle, is not to be preferred over that of the average milk of large herds properly bottled before being sent to market. So likewise as to the composition of the "strippings" of the udder. They are not usually procurable, and their greater richness in fat and deficiency in casein can be better arrived at, even when ordinary whole milk is used, by appropriately modifying its composition.

Limiting our consideration strictly to commercial bottled milk, it becomes of the greatest importance to inquire into the present conditions regulating its production and handling at the farm, during transit, and in delivery to the consumer. Hitherto, these conditions have fallen far short of the requirements which chemical and medical science should rightly impose upon milk as the prime article of artificial infant nutrition. The State laws have checked the adulteration of milk by addition of water and removal of cream, but as yet have done little, and that only incidentally, in the way of guaranteeing its wholesomeness and improving its quality. In fact, enlightened public sentiment, assisted and directed by the medical profession, will do more in this direction than can be expected at present from the State. And the same remark is true of the efforts of the dairyman. What is being done and should be done is best exemplified by a recital of the provisions of a legal contract drawn up between a committee of certain medical societies in the vicinity of New York on the one hand and a competent dairyman on the other. The latter undertakes that his herd of Holstein and Jersey cattle shall be regularly and frequently inspected by a veterinarian selected by the committee and paid by the dairyman. All cattle that are pronounced by the surgeon, for any cause whatsoever, disqualified to produce pure sound milk are forthwith excluded from the herd. Interbreeding more frequently than the fourth generation is interdicted. The cattle must be kept in a large, well-ventilated, well-lighted stable, with ample space and no overcrowding, with abundance of pure water for drinking and cleansing; with perfect drainage; with dry cemented floors; with clean fresh bedding of hay; and with arrangements for securing them in the stall which shall give ample liberty to the movements of the head and for lying down, but shall do away with the necessity of chains or other fastening. Separate stalls and partitions, as interfering with ventilation and cleanliness, are done away with. The cow-stables must be removed from those in which horses, chickens, and other stock are kept by so great a distance that the cattle can in no wise come in contact with the other animals. The cows must be groomed daily, and the teats washed before each milking. The milkmen must perform their own toilets before milking, being especially required to thoroughly cleanse their hands and to remove the dirt beneath the finger-nails, wearing also unsoiled clothing. The feeding is to be regulated by the season in such

wise that the milk produced shall conform to the highest feasible standard of excellence. Abundance of wholesome pasture, hay, meal, fodder, and ensilage is demanded, but the refuse of glucose-factories, brewers' grains, swill in any form, etc. are interdicted. There are also provisions in the contract that the cattle shall not be worried, heated, or driven, or milked except after proper interval after calving. The milking must be done with scrupulously cleansed vessels; the milk filtered through fine metallic gauze, then cooled in a dust-free atmosphere in such wise as to lower the temperature as rapidly as possible, and also to permit the escape of the gases along with the animal heat; and, finally, transferred to bottles rendered as nearly sterile by cleansing with boiling water and steam as possible. These jars, which must be entirely full, are closed by a metallic cover, sealed, transferred to boxes with a layer of ice on top of them, and delivered at an early hour in the day, the temperature of the milk never being allowed to rise in the interval above 50° F. The dairyman further undertakes to pay for the services of a competent chemist and biologist, who shall frequently test the milk, and whose analyses and certificates shall accompany it. He also undertakes to have his stables, cattle, feed, bottling arrangements, etc. open at all proper times to inspection, and to comply with all other requirements of the committee which they in their judgment shall deem essential to securing the highest attainable degree of quality and purity. The only obligation which the committee assumes is that it permits the milk to be sealed with a label bearing the name of the dairy and the dairyman, and the legend "Certified Milk," and to be accompanied by the certificate of purity bearing the name of the committee, the chemist, biologist, and veterinarian.

Milk in the human gland or cow's udder, when tuberculosis or kindred disease is absent, contains no bacteria. Indeed, by rejecting the first portions and excluding floating particles in the air, sterile cow's milk can be obtained, and contrivances to this end have been patented; but they are quite impracticable. So likewise is the proposition to sterilize all the milk before it leaves the farm by heating it at 230° F. for a sufficient length of time completely to destroy every spore which might by any possibility be present. Consumers would not pay for the skill, time, and apparatus required, and the process itself produces unfavorable changes in the milk. The first portion of this objection applies also to the proposition that the milking should be done directly into sterilized bottles, and the milk then Pasteurized by heating to a temperature of 160°–170° for twenty minutes.

Any of the bacteria present in the air, water, ground, or derived from the diseased or filthy condition of those who handle the milk at any time, or arising from the animals themselves, may possibly find their way into milk. And, inasmuch as this fluid is an excellent culture-medium, they multiply with great rapidity. But these things demand suitable care for their prevention, and not a care involving the compulsory sterilization of all milk. The author believes that no more should be required of the dairyman than the reasonable precautions above detailed, which self-interest also demands. Then a false security will not be placed in legal requirements sure to be evaded or neglected, and necessitating an army of skilled inspectors, veterinarians, and chemists to enforce. The few ounces of milk needed for artificial nursing are best sterilized immediately before use, and this is best done in the course of the preparation essential to adapt it for infant feeding, either just before transfer to the bottle or in the bottle itself. By so doing, the fact, usually lost sight of, will be kept constantly in mind—that the same precautions as to the bottle, nipple, the water used, the exclusion of floating particles from the milk, and the keeping of it in a refrigerator are as essential to preserving the sterility of the milk as

its sterilization in the first instance. Washing in boiling water cannot be trusted to remove the adherent skin of fat and casein on the milk-vessels; some soda must be used; the rubber nipple should be turned inside out over the finger and scrubbed with a brush and precipitated chalk.

Supposing that the present enlightened public sentiment has secured such a legalized system of sanitary cattle-inspection and milk-control as to make the reasonable precautions now exercised voluntarily by honorable dairymen obligatory upon all, bottled milk, which I shall term "sound dairy milk," presents the following characteristics: In color it varies from white to yellow. Even when allowed to fall in drops from the end of a rod it exhibits a dense white opacity and consistency, the fluidity and bluish-white color of watered or inferior milk being absent. It is almost neutral, reddening litmus-paper very feebly. On standing, the cream rises in the neck of the quart bottle commonly used until it forms a layer about two and a half inches in depth. These physical characters are all that need be noted. If they are absent, if the milk is thin and watery, if it has a bluish, blue, strong yellow, or red color, if it is stringy, lumpy, or glutinous, if it has a flat, stale, sour, or any abnormal taste or odor,—it is simply to be rejected, and its investigation left to the milk inspector and chemist.

Many analyses of such bottled milk afford me the following average results, which are given as preliminary to the still better figures that will come with "certified milk:"

Fats	3.75	per cent.
Lactose (milk-sugar)	4.42	"
Albuminoids	3.76	"
Ash	0.68	"
Total solids	12.61	per cent.

In some of the States the legal standard calls for 12.5 per cent. of total solids and 3 per cent. of fat. It is much to be deplored that in other States, as in New Jersey, the standard demands only 12 per cent., and unless the fat falls below $2\frac{1}{2}$ the milk is assumed to be unskimmed. It was made thus low in order that no lack of care in housing and cleanliness, no inadequacy of feeding, no abstraction of cream from the evening milk (half-skimming), and no accidental or judicious watering should bring the owner or vendor under condemnation of law. For the same reasons it is assumed that any milk which has a higher specific gravity than 1.029 at 60° F. (100° on the lactometer scale) is pure, whereas the average of sound dairy milk should be 1.0297.

Human Milk.—Having given the above general characteristics of cow's milk, it is necessary to do the same for human milk, and then proceed to a more specific comparison of their resemblances and differences. And in the first place, while all the conditions and environment are arranged to develop the milk-secreting function of milch cattle, in the human family, on the other hand, they are more and more ignored as women become burdened with the increasing duties and dissipations of modern society. The regular life with moderate enjoyments, exercise, and occupation, the simple nourishing diet, with abundance of fresh air and rest, which are most favorable to the milk-secretion, are sacrificed, with the result of arresting or diminishing the flow and deteriorating the quality of the milk. Stimulants, narcotics, improper or highly-seasoned food, functional disorders with their attendant medicines, violent emotions and paroxysms of grief, anger, and pain, render the milk unwholesome and sometimes dangerous. As a contribution to the chemistry

of this subject I give in an accompanying table the results of 80 analyses of samples of milk obtained from women of different nationalities, age, stage of lactation, and physical constitution, but all living in a lying-in hospital under the same conditions and eating the same food. (See pp. 42 and 43.)

The analyses are arranged according to the period of lactation, except in cases where several samples were taken, these following consecutively. Many hundred analyses would be required to determine what differences, if any, are due to nationality or to the physical characteristics of the mother—whether black, blonde, or brunette, or, more minutely, the color of the eyes, hair, complexion, etc. But the influence of the physical condition was pronounced, the best milk not coming from women of robust habit (Column I.), but from those whose nourishment appeared rather in the milk-secretion than in the fattening of the mother (Column II.):

	I. (6 cases).	II. (6 cases.)
Fats	3.71	3.96
Lactose	6.94	6.74
Albuminoids	1.44	2.12
Ash	0.25	0.22
Total solids	12.34	13.04

The reaction of every sample was alkaline, the alkaline reaction persisting during one or more days. The color varied from bluish-white through chalky-white to strong yellow, but the color was not a necessary index of the composition: the milk of a German (No. 34), which was the richest in fats (6.89 per cent.), lactose, and total solids, was chalky-white in color, while that of another German (No. 8), which was yellow, was very low in fats, having only 2.31 per cent. Though the amount of lactose is more than a third greater than in cow's milk, yet the taste can hardly be called sweet, and while the total solids (13.27) and the specific gravity (1.0313) are both higher than in cow's milk, yet the consistency is much thinner. This is due to its much smaller content of albuminous matters, more especially of the caseinous or cheesy material.

The average amount of nitrogenous matters (albuminoids) is somewhat greater at beginning of lactation, but the difference is not very marked. In truth, the feature brought out by this long series of analyses, which overshadows every other in significance, is the fact that there is no progressive change in the composition of milk during lactation, but after the function has been normally established the milk remains substantially the same during the entire period. This is what might be anticipated from what much larger experience teaches in regard to cow's milk, but it is at variance with notions commonly entertained, and which have led to elaborate and utterly useless diets for infant nutrition. The child obtains more nutriment day by day, but it is by spontaneously increasing the quantity according to the best rule, which is that of normal appetite, and not by absorbing "stronger and stronger food."

Comparison of Cow's Milk and Human Milk.—Before proceeding farther, the general characteristics may advantageously be summed up in the following comparison:

	Sound Dairy Milk.	Woman's Milk.
Reaction	Feebly acid.	Persistently alkaline.
Specific gravity	1.0297	1.0313
Bacteria	Always present.	Absent.
Fats 3 6	—average, 3.75 2	—average, 4.13
Lactose 3.5 to 5.5—	4.42 5.4	to 7.9— " 7.0
Albuminoids . 3 6	— " 3.76 0.85 to 4.86—	" 2.0
Ash 0.6 to 0.9—	" 0.68 0.13 to 0.37—	" 0.2

TABLE OF ANALYSES OF SAMPLES OF MILK FROM WOMEN OF DIFFERENT NATIONALITIES, AGE, ETC.

1.	2.	3.	4.	5.	6.		7.	8.	9.	10.	11.	12.	13.	14.
No.	Mother's age.	Nationality.	Color of hair.	Period of lactation.	Breast.		Interval since nursing.	Color of milk.	Specific gravity.	Albuminoids.	Lactose.	Fat.	Ash.	Total solids.
13	28	German . . .	Brown . . .	1 day.	. . .	L.	2 hours.	1.030	2.40	6.45	6.01	0.22	15.08
14	28	German . . .	Dark brown .	1 "	. . .	L.	2 "	1.032	2.52	6.44	4.95	0.27	14.18
15	19	Irish . . .	Dark brown .	2 days.	. . .	$\frac{1}{2}$ L.	5 "	1.034	3.12	6.47	5.49	0.32	15.40
18	7	Nos. 7-35, same mother.	6 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	. . .	Dull white .	1.033	3.95	7.92	4.37	0.22	16.46
28	28	19 "	$\frac{1}{2}$ R.	L.	. . .	White . . .	1.031	2.23	7.39	2.95	0.21	12.78
35	35	29 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	. . .	White . . .	1.032	2.43	7.34	3.13	0.21	13.11
47	26	American . . .	Brown . . .	2 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	Yellow . . .	1.032	4.86	5.40	3.36	0.20	13.82
6	33	Pole . . .	Dark brown .	2 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	3 "	Yellowish-white.	1.030	1.45	7.24	3.20	0.24	12.13
38	20	German . . .	Brown . . .	2 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	3 "	White . . .	1.021	1.82	6.96	3.97	0.19	12.94
15	22	German	3 "	. . .	L.	2 "	1.032	2.18	6.75	2.84	0.13	11.90
16	16	5 "	. . .	L.	. . .	Yellow . . .	1.030	0.85	5.50	6.16	0.22	12.73
26	16	No. 15 to No. 30, same mother.	Light, a typical blonde.	13 "	. . .	L.	2 "	1.030	2.08	6.98	3.38	0.20	12.54
29	29	17 "	. . .	L.	. . .	White . . .	1.030	1.81	6.88	2.80	0.20	11.69
30	30	27 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	. . .	Yellowish-white.	1.031	2.11	7.41	5.04	0.20	14.76
40	24	Negress . . .	Black . . .	3 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	20 min.	Yellow . . .	1.032	1.75	6.94	3.68	0.28	12.65
64	24	3 "	. . .	L.	30 "	1.0312	2.00	6.69	3.96	0.32	12.97
8	29	Hungarian . .	Black . . .	4 "	R.	. . .	4 $\frac{1}{2}$ hours.	1.030	2.15	6.51	2.31	0.28	11.25
45	22	German . . .	Light brown .	5 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	Yellow . . .	1.031	2.33	7.48	2.47	0.16	12.44
22	22	American . . .	Brown . . .	5 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	30 min.	Yellowish-white.	1.032	1.96	7.28	4.74	0.30	14.28
67	28	Scotch . . .	Fair . . .	6 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	30 "	1.0322	2.45	6.08	3.82	0.19	12.54
23	23	Irish . . .	Black . . .	6 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	30 "	White . . .	1.031	0.22	13.01
21	18	English . . .	Black . . .	8 "	. . .	L.	1 hour.
32	28	German . . .	Dark brown .	10 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	0 min.	Yellowish-white.	1.030	1.53	5.84	5.62	0.14	13.13
34	23	German . . .	Brown . . .	10 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	Chalky-white .	1.034	2.19	7.46	6.89	0.25	16.79
33	23	Irish . . .	Dark brown .	11 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	0 "	Yellow . . .	1.030	2.24	6.25	2.76	0.35	11.60
65	23	Dane . . .	Light . . .	12 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 "	1.0319	2.25	7.12	5.85	0.15	15.37
4	23	Irish . . .	Dark brown .	13 "	$\frac{1}{2}$ R.	L.	3 hours.	1.0346	1.73	7.25	2.95	0.18	12.11
39	23	Irish . . .	Brown . . .	13 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	0 "	Yellowish-white.	1.030	2.33	5.78	4.21	0.21	12.53
48	22	American . . .	Red . . .	17 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	Yellowish-white.	1.032	1.93	6.95	5.59	0.18	14.65
31	20	German . . .	Dark brown .	19 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ hour.	Chalky-white .	1.033	2.27	6.75	5.96	0.15	15.13
24	20	Italian . . .	Black . . .	20 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ "	1.030	1.94	7.45	3.61	0.16	13.16
9	29	Irish . . .	Brown . . .	22 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 hours.	1.032	2.05	7.08	3.00	0.13	12.26
51	16	American . . .	Dark brown .	23 "	. . .	L.	2 "	Yellow . . .	1.030	2.42	6.95	5.60	0.19	15.16

	German.	Light brown.	23 days.	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	2.17	7.44	4.36	0.29	14.26
22	German.	Brown . . .	25 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{4}$ hours.	1.0317	7.44	4.36	0.29	14.26
3	Irish . . .	Brown . . .	26 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	6 "	1.0353	7.23	4.12	0.18	14.07
5	Irish . . .	Dark brown.	27 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.030	7.23	2.12	0.15	11.02
21	Irish . . .	Dark brown.	30 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ hour.	1.0351	7.53	3.55	0.21	12.97
25	German . . .	Brown . . .	41 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ hour.	1.0307	7.07	2.73	0.22	11.13
12	Irish . . .	Brown . . .	45 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.031	7.07	2.44	0.20	11.37
23	German, same as 1	Brown . . .	49 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0321	7.20	5.58	0.17	14.39
19	German . . .	Light brown.	45 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	3 "	1.034	7.37	5.02	0.17	14.05
18	German . . .	Light brown.	46 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	3 "	1.031	6.99	3.06	0.20	12.23
30	Slavonic	Brown . . .	50 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	1.030	6.61	4.02	0.30	14.08
20	Bohemian.	Brown . . .	53 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	1.030	7.19	2.11	0.20	12.93
26	Bohemian.	Brown . . .	82 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 hours.	1.030	7.19	2.11	0.19	10.92
10	Scotch . . .	Brown . . .	88 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.029	6.83	4.28	0.37	13.30
53	Irish . . .	Red . . .	88 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	7.00	2.41	0.21	11.63
27	German . . .	Light brown.	89 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ hour.	1.031	6.76	6.78	0.16	15.85
28	German . . .	Light . . .	90 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.030	6.76	6.78	0.16	15.85
52	Swedish . .	Light . . .	93 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.030	6.57	4.94	0.27	14.21
25	German . . .	Brown . . .	93 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	3 "	1.026	6.57	4.94	0.27	14.21
55	Irish . . .	Brown . . .	115 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.032	7.00	5.81	0.22	15.22
20	Irish . . .	Brown . . .	115 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.032	7.00	5.81	0.22	15.22
19	Irish . . .	Dark brown.	126 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	1.030	7.34	3.10	0.15	12.34
23	German . . .	Dark brown.	132 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.030	7.34	3.10	0.15	12.34
30	American . .	Brown . . .	150 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.031	7.32	3.77	0.18	12.77
43	American . .	Brown . . .	150 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	7.32	3.77	0.18	12.77
42	Irish . . .	Brown . . .	167 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	7.38	4.16	0.25	13.76
22	Swedish . . .	Brown . . .	180 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	$\frac{1}{2}$ hour.	1.030	7.31	4.34	0.21	12.74
33	Swedish . . .	Light . . .	180 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.031	7.31	4.34	0.21	12.74
25	American . .	Light . . .	180 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.030	7.31	4.34	0.21	12.74
44	American . .	Black . . .	186 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.031	7.24	4.09	0.31	13.35
46	Mulatto . . .	Black . . .	186 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.030	7.24	4.09	0.31	12.99
24	Mulatto . . .	Light brown.	217 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	6.39	4.75	0.22	13.42
50	American . .	Light brown.	270 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	6.39	4.75	0.22	13.42
19	Irish . . .	Light brown.	271 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.031	6.39	4.75	0.22	13.42
25	Scotch . . .	Light brown.	270 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 min.	1.031	6.95	4.61	0.21	13.80
60	Irish . . .	Light brown.	271 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 min.	1.031	6.95	4.61	0.21	13.80
28	German . . .	Brown . . .	12 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0296	7.02	3.85	0.22	13.04
61	Irish . . .	Brown . . .	12 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0296	7.02	3.85	0.22	13.04
62	Irish . . .	Black . . .	27 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0296	7.02	3.85	0.22	13.04
63	Irish . . .	Light brown.	19 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0296	7.02	3.85	0.22	13.04
22	Irish . . .	Light brown.	19 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	5 "	1.0296	7.02	3.85	0.22	13.04
56	French . . .	Dark brown.	210 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	1 hour.	1.0297	7.41	4.74	0.21	13.52
57	Irish . . .	Brown . . .	90 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.0297	7.41	4.74	0.21	13.52
58	Irish . . .	Dark brown.	153 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 hours.	1.0297	7.41	4.74	0.21	13.52
59	Irish . . .	Light brown.	92 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
36	Irish . . .	Light brown.	92 "	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
37	Irish . . .	Light brown.	lost . . .	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
		Descriptions	lost . . .	$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52
				$\frac{1}{2}$ R.	$\frac{1}{2}$ L.	2 "	1.0297	7.41	4.74	0.21	13.52

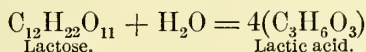
On an average, human milk has about $\frac{1}{2}$ of 1 per cent. more fat than average sound dairy milk, and $2\frac{1}{4}$ per cent. more lactose. On the other hand, it has $\frac{1}{2}$ of 1 per cent. less mineral matter, and, what is most important, *but 2 per cent. of albuminoids, or about half the quantity in cow's milk.* The fat is the most variable constituent, as is the case in cow's milk also. But in both the sum of all the other constituents besides fat is a nearly constant quantity, amounting in the vast majority of samples to about 9 per cent. The significance of this physiological fact must not be lost sight of. It shows that the final tendency and result of the complicated metabolic changes, which take place in the protoplasmic cells of the mammary gland, is to secrete a nearly constant total amount of nitrogenous, carbohydrate, and saline material, while allowing the secreted fat to exhibit a wide and independent variability. An increase in the amount of nitrogenous food does not increase the nitrogenous element in the milk secreted by a nursing woman beyond the general limit implied in the above rule, the metabolism in this case resulting in an increase of the fat. An excess of fat, on the other hand, diminishes the metabolism. And, as a practical deduction from the above, there results the necessity of feeding a nursing woman on a diet which shall contain a sufficiency of proteid matters, but not on a rich food, the former yielding by transformation not only the albuminoids, but also the fats and lactose of the milk, whilst the latter may not in this sense be nourishing, and may impair the metabolic activity whereby the due proportion of the various constituents of the milk is normally maintained.

It is necessary to the further understanding of the problem of infant nutrition, and especially of artificial feeding, to study in detail the similarities and differences of the individual constituents of woman's and cow's milk.

Lactose.—The lactose in the two secretions is chemically, physically, and physiologically identical. The statements based on clinical results to the effect that the lactose of cow's milk exerted a peculiar diuretic action and produced glycosuria and set up abnormal digestive fermentations, etc. will have to be reviewed. Until very recently all the samples of lactose coming into my laboratory, even those supplied by manufacturers of highest repute as chemically pure, were far from being so. They contained residues of the proteids of milk and spores, the taste, appearance, and properties of the lactose being thereby altered. So great is the present use of lactose in medicinal preparations that correspondingly great improvements have been made in its manufacture, resulting in the production of a very pure, hard, white, transparent, crystalline substance.

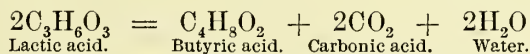
The carbohydrate element, which is made up of starches, the many varieties of sugar, etc. in the food of adults, and which constitutes the largest part of most vegetables and fruits, is represented in milk by lactose only. This body is intermediate in its chemical properties between cane-sugar and starch, being, like the former, soluble, but with a taste hardly perceptibly sweet. Its main function is to supply by oxidation the animal heat, and, inasmuch as the human infant cannot maintain its animal heat by locomotion, and yet at the same time this heat must be preserved at even a higher temperature than that of the adult, the lactose is relatively the largest constituent of human milk, forming more than one-half its total solid matter. Being already in a soluble condition, it is directly assimilable, and, unlike starch, requires little or no expenditure of energy to effect its transformation prior to digestion. Under the influence of certain bacteria, acting as ferments, the lactose is decomposed, with the formation of lactic acid. Up to the present time ten varieties of bacteria, including, along with the *bacillus acidi lactici*, certain species of micrococci and sphæro-

cocci, have been described as more especially concerned in the lactic fermentation of milk. They all bring about the curdling of the milk, but some of them at the same time give rise to the formation of gas and alcohol, and others do not. The primary change is due to the simple splitting of the molecule of lactose into four molecules of lactic acid by addition of a molecule of water:

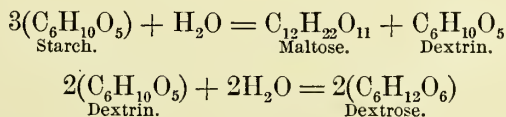


This change, which is the ordinary normal one, ensures the curdling and the development of lactic acid initiative to milk digestion. Under the influence of other ferments the molecule of lactic acid may break up into a molecule of alcohol and carbonic acid ($\text{C}_3\text{H}_6\text{O}_3 = \text{C}_2\text{H}_6\text{O} + \text{CO}_2$), but this decomposition is secondary and abnormal, and takes place less readily and more slowly than the decomposition of grape-sugar, glucose, or dextrose into alcohol and carbonic acid under like circumstances.

Besides this fermentation, which results in the separation of a curd by means of lactic acid, there is another fermentation, which is accompanied by the development of a neutral or alkaline reaction. In this case the curd first formed may all eventually pass into solution, being converted into soluble peptones. The bacteria giving rise to these changes originate two soluble substances acting as ferments, one acting like rennet to curdle the milk, the other dissolving the curd and exerting a peptonizing action. There is also produced leucin, tyrosin, ammonia, and, more especially, butyric acid, which last body gives its name to this kind of fermentation. Artificially, it is induced by contact with putrid cheese. In the digestive tract the butyric fermentation is usually brought about by the prolonged stay in the bowels of the undigested curds of milk or of a foreign irritant substance like starch, or by both. It is essentially a process of putrefactive decomposition, not present in normal digestion. In its simplest form the change may be represented by the formula



While starch is the principal carbohydrate of adult food, it cannot properly be used in infant feeding on account of the absence of the ferment essential to its digestion. This starch-digesting ferment exists under the name of ptyalin in the saliva, and also is present to some extent in the pancreatic juice, but its amount in infants is very small, and its secretion is not established until after the third month. By its action the starch is made to take up a molecule of water and then decompose into maltose and dextrin, the latter body, by continuance of the same action, passing into dextrose; thus:



Liebig proposed to effect this change by means of the diastase contained in malt, and his suggestion has been extensively followed. But the objection still remains that the saccharine substances thus produced, like the vegetable sugars in general, are not the same carbohydrate which is normally present in milk, and it has not as yet been satisfactorily established that they undergo in digestion the same series of changes and oppose equal resistances to abnormal fer-

mentation. Though cane-sugar or sucrose, malt-sugar or maltose, and milk-sugar or lactose, all belong to the same general class of sugars known as saccharoses, with the formula $C_{12}H_{22}O_{11}$, yet their physical and chemical properties are essentially different, and so also their behavior when in presence of certain ferments.

Fat.—So far as is known at present, the principal difference between the fat-globules of woman's and cow's milk is in the relatively greater size of the former, which vary between 0.001—0.02 mm., while the latter average 0.00014—0.0063 mm. in diameter. The assumption that each globule is surrounded by a membranous envelope has been disproved, the finely-divided fat existing as naked globules, on the surface of each of which a number of albuminous molecules are condensed by molecular attraction, and the coalescence of the fat particles thereby hindered.

Albuminoids.—While the lactose of human and cow's milk is identical, and the fats are very similar, the nitrogenous portion presents so many and important differences that the question of the successful substitution of cow's milk for human principally depends upon whether or no these differences can be compensated or overcome. In both secretions the nitrogenous portion consists mainly of casein and lactalbumin. In addition, there are substances of the nature of peptones, in small quantities, but to what extent they exist naturally, and to what degree, in the case of cow's milk, they are formed by the peptonizing action of bacteria, is not at present determined. Casein is an acid body existing in milk in combination with alkali, forming principally potassium caseinate. But the reactions of this body are complicated by the presence of other mineral bodies, and more especially of calcium phosphate. When dilute acid is added the casein of cow's milk readily precipitates in coarse coagula or clots, but that of woman's milk requires more acid for its precipitation and separates not in lumps, but in a fine powder which dissolves in excess of the acid. The lactalbumin remains in solution in the whey after separation of the casein. By boiling it is rendered insoluble. It closely resembles serum-albumin. While in cow's milk the total fraction of the albuminoids precipitable by acid (casein) exceeds by about four times the non-coagulable portion, in human milk these proportions are reversed, the non-coagulable part being about twice the coagulable portion. Similar differences exist in the coagulum formed by the acid gastric juice: in the one case an excess of insoluble cheesy masses, in the other a relatively small amount of finely divided soluble flakes, being formed. Taking equal weights of the two secretions, the coagulum of woman's milk is but one-fifth as much as that of cow's milk. The comparative smallness of this quantity must be as carefully considered as the difference in the compactness and solubility of the coagula themselves. It explains the rapidity with which infant digestion is overtaxed even by small amounts of undiluted cow's milk.

Inorganic Matter.—The mineral matter in cow's milk is more than three times that in woman's milk, and especially great is the excess of calcium phosphate, which is four times larger. This excess is due to the correspondingly larger amount of casein in cow's milk, with which substance the calcium phosphate and the potash are principally combined. The soda appears to exist in solution along with the lactalbumin as common salt. It is noteworthy that the lime is already relatively greater in the cow's than in human milk, and it is open to serious question whether the practice of using cow's milk alkalinized by excess of lime is as desirable, in the case of normal digestion, as it was thought to be before the composition and properties of the constituents of milk were known. The following table presents the relative composition of

the ash of cow's milk (Fleischmann) and of woman's milk (König), and also the percentages of each constituent (Bunge):

	Cow's Milk.		Woman's Milk.	
Potash	24.5	0.18	33.78	0.07
Soda	11.0	0.11	9.16	0.03
Lime	22.5	0.16	16.64	0.03
Magnesia	2.6	0.02	2.16	0.01
Oxide of iron	0.3	0.0004	0.25	0.0006
Phosphoric acid	26.0	0.2	22.74	0.05
Sulphuric acid	1.0	—	1.89	—
Chlorine	15.6	0.17	18.33	0.04

II. THE CHEMISTRY OF ARTIFICIAL FOODS.

Two methods have been followed in the attempt to solve the problem of artificial feeding. The easier, and that most generally adopted, which would also appear to be the more natural method, is that of attempting to produce a food which should resemble as closely as possible woman's milk. The other method aims to produce a food or foods which should be especially adapted to the demands of nutrition for each particular infant in health or disease: it is open to great diversities of opinion, due to opposing clinical experiences, and is adapted rather to the treatment of special cases of disordered digestive and other functions than to common use. By general consent the advocates of the first method have selected cow's milk as the basis upon which to build. The difficulty of obtaining cheaply, readily, and of proper quality the milk of the ass, the goat, or of any other animal than the cow, has rendered the discussion of the possible advantages of such milk quite useless.

Dilution.—The first expedient in connection therewith was that of dilution with water until the percentage of albuminoids and salts should approximate to that in woman's milk. But no amount of dilution with water alone is adequate to prevent the separation of the curd in coarse, indigestible lumps in presence of the acid secretions of the stomach. The next device was the addition of an excess of lime-water, so as to partly neutralize the gastric juice and allow much of the milk to pass unchanged from the stomach and undergo digestion in the bowels. As the chemistry of the milk salts indicates, the excess of lime is abnormal, and its addition is an expedient to meet a therapeutic condition connected with an over-development of acidity, and not to change the nature of the difficultly digestible casein itself.

Predigestion.—To effect this latter change previous digestion with dilute acid and pepsin was resorted to, and latterly this gave place to the more successful digestion with pancreatin in alkaline solution. Both methods were confined to cases of greatly impaired digestion, and the predigestion was carried as far as possible. But inasmuch as in woman's milk there naturally remains about one-fifth of the albuminoids in a caseinous condition, the most recent practice is that of using a limited amount of pancreatin, acting for so short a period that the process shall initiate the peptonization, and then be arrested by the destruction of the ferment. The casein is thereby left in such a condition that it separates on acidifying as a fine white powder, while the biuret reaction for the albuminates becomes strongly developed.

Sterilization.—Recently the fact that woman's milk contains no bacteria, while cow's milk usually contains large numbers and many kinds, pathogenic species possibly included, has been strongly insisted upon. To overcome this objection the practice of sterilizing the milk by repeated heating to a temperature above the boiling-point of water has been extensively followed.

So far as the destruction of all bacteria and their spores is concerned, the process is successful, but the clinical results which have attended the use of such sterilized milk have revealed serious drawbacks. It prevents the spread of zymotic diseases through the medium of milk; it is efficacious in checking many gastro-intestinal disorders; but its continued use is accompanied by a failure to afford adequate nutrition. Besides the destruction of the bacteria, the prolonged heating to or above the boiling-point brings about other changes which are in the nature of deteriorations. More especially the lactalbumin loses its solubility, and the fat-globules are made to coalesce with one another and some of the insoluble albuminous matter. For these reasons the application of continued heat in the process of sterilization is inadvisable, and is now being discontinued.

Sterilization at a Low Temperature (Pasteurization).—In this process of preparation the milk is kept for a brief interval, ten to twenty minutes, at a temperature of 160° – 170° F., or raised during heating continued for ten minutes just to the boiling-point. While this process will not destroy all the germs which are in the form of spores, it will destroy the spores of tuberculosis, scarlet fever, pneumonia, and typhoid, and almost completely inhibit the existence of the developed spores, or bacteria, of every kind.

Pasteurization with Partial Predigestion (Humanized Milk).—The adjustment of the lactose and the bringing about of a permanently alkaline reaction are effected by the presence in the diluted sterilized milk of such an amount of lactose and of the alkaline milk salts as will effect this result. In order to raise the percentage of fat to that contained in woman's milk, cream may be added, or some vegetable oil like olive or cocoa, or animal oil like that of cod-liver. At present, by the aid of the Leval separator, cream has become a commercial article easily obtained, and its use is more convenient and better understood than that of the other fat substitutes, which require to be further investigated. Inasmuch as it contains some casein and bacteria, due allowance must be made for both in the process of modification heretofore explained. In practice, by the use of a preparation of pancreatin, lactose, and alkaline milk salts originated by Fairchild Brothers & Foster of New York, and known as "Peptogenic Milk-powder," the author has found that with ordinary bottled milk, cream, and water a modified sterilized milk is obtained which corresponds so closely to woman's milk that he has given it the name of "humanized" milk. The proportions recommended are—

Milk	$\frac{1}{2}$ pint.
Water	$\frac{1}{2}$ pint.
Cream	4 tablespoonfuls.
Peptogenic Milk-powder	1 large measure.

The mixture is heated on a hot range or gas-stove with constant stirring, the heating being so conducted that at the end of ten minutes it is brought to the boiling-point. The temperature of 160° to 170° is high enough to destroy the ferment, and this temperature, continued for twenty minutes, kills the bacteria also. But it is so much easier to quickly raise the temperature for a moment to the boiling-point, which also effects both objects, that the latter method is to be preferred when by a process of partial peptonization, as in the process described, the main portion of the albuminoids has been brought to a permanently soluble form.

The milk thus prepared is slightly alkaline and sterile. It contains, according to the author's analyses, bottled market milk being used in its preparation, the following proportions of constituents:

Fat	4.5 per cent.
Albuminoids	2.0 “
Lactose	7.0 “
Ash	0.3 “
Total solids	13.8 per cent.

When lime is used to counteract not only the slight acidity of market milk, but also with the object of forming a soluble calcium caseinate which will not be decomposed by the acid of the gastric juice and curds of casein thereby precipitated, the lime must be added in considerable quantities. A mixture of 2 ounces of milk, 2 ounces of lime-water, and 2 ounces of cream, to which a teaspoonful of sugar of milk has been added, contains only a grain of lime, a quantity too small to effect any notable chemical change of the casein. If this mixture is sterilized, it should be done at a temperature between 160° and 170°, since heating to the boiling-point causes some decomposition of the albuminoids in presence of alkali.

“**Condensed Milk.**”—When condensed milk is used the preceding remarks require to be somewhat modified on account of the different modes of preparing this substance. This will be readily understood by comparing the composition of (I.) milk condensed with added cane-sugar, mean of forty-one analyses; (II.) the same diluted with eight times its weight of water; (III.) Anglo-Swiss milk, preserved without added sugar; (IV.) American-Swiss, preserved; (V.) No. III. diluted with five times water.

	I.	II.	III.	IV.	V.
Fat	12.10	1.51	13.21	11.55	2.64
Albuminoids	16.07	2.01	11.36	14.10	2.27
Lactose	16.62	2.08	15.29	13.04	3.05
Sucrose	22.26	2.78			
Ash	2.61	0.32	1.78	2.09	0.36
Total solids	69.66	8.70	41.64	40.78	8.32
Water	30.34	91.30	58.36	59.22	91.68

When largely diluted with water, so that the percentage of albuminoids is approximately the same as in human milk, the fat and lactose are brought far below the quantity proper for infant nutrition. Nor is the deficiency adequately supplied by the added sucrose of the milks condensed with this substance. Referring to these points, Dr. Louis Starr justly remarks: “Condensed milk is frequently recommended by physicians, and largely used by the laity on their own responsibility. It keeps better than cow’s milk, and is supposed to be more readily digested by infants. The latter supposition is a mistaken one, and arises from the overlooked fact that condensed milk is always given dissolved in a large proportion of water, while cow’s milk is too frequently used insufficiently diluted or otherwise improperly prepared. The author is convinced of the accuracy of this statement from a number of years’ close study of the subject. Condensed milk contains a large proportion of sugar, forms fat quickly, and thus makes large babies; sugar also counteracts the tendency to constipation—often a troublesome complaint in hand-feeding. These advantages are unquestioned, and, together with the ease of preparation, are those which place it so high in the esteem of monthly nurses. It is equally true, however, that as a food it does not contain enough nutrient material to supply the wants of a growing baby. . . . It must be remembered also that condensed milk, when long kept or when packed in imperfect cans, not infrequently undergoes decomposition, and thus becomes utterly unfit for use.”

Attenuation.—An entirely different method of increasing the digestibility

of the casein is that of adding farinaceous or gummy substances, the action of which is not chemical, but mechanical, and depends upon the separation which they effect of the otherwise cheesy masses into a multitude of fine particles. Experiments in the laboratory of the author showed that when diluted cow's milk, to which a solution of cane-sugar, grape-sugar, barley-water, starch-water, or gelatin had been added, was treated with acid, the precipitated casein carried down with it from one-third to more than twice its weight of the added substance. Gelatin more especially must be used in very small quantity, since otherwise it entirely arrests the precipitation of the casein. One of the simplest and best of these attenuants is barley-water, added to one-third its volume of milk. It may be prepared by boiling two teaspoonfuls of pearl barley in a pint of water in an open saucepan until the bulk is reduced to two-thirds, and then straining. Instead of barley, oatmeal may be used, or gelatin. To prepare the latter put a piece of plate gelatin an inch square into a half-tumblerful of cold water, and let it stand for three hours; then turn the whole into a teacup, place this in a saucepan half full of water, and boil until the gelatin is dissolved. When cold this forms a jelly: two teaspoonfuls are sufficient to thicken a mixture of three ounces of milk and five of water.

Dextrinized Attenuants.—A gummy material like dextrin, or a gelatinous substance, or a saccharine body, or a finely-divided starch like that occurring in barley- or oatmeal-water, along with more or less glutinous extractive matter, is far better adapted to serve mechanically as an attenuant of the coagulated casein than farinaceous foods in their ordinary condition. Many different preparations are sold in which, by prior heating (dextrinizing) or by digestion with diastase, wheat and barley flours are modified to this end. By the action of heat at 300° to 400° the principal substance which is formed is dextrin, a body differing from starch by its being soluble and by having the physical characters of a gum. Diastase produces principally maltose along with dextrin. The flour selected for either treatment should be highly albuminous, made of wheat grown at certain seasons and of certain grades, and should be the best grade of that made by the roller process. Grouping together under the head of soluble carbohydrates the sucrose, dextrose, maltose, and dextrin originally present or made by treatment, the changes can be traced in the following table. The first column gives the composition of a wheat flour, the second the same after baking. The remaining columns exhibit similar products from other specimens of wheat flour, the process having been carried further in some of the dextrinized foods than in others :

	Wheat flour.	Same baked.	Blair's Wheat Food.	Imperial Granum.	Ridge's Food.	Schuma- cher's Food.
Water	9.02	7.78	9.85	5.49	9.23	6.26
Fat	1.01	0.41	1.	1.01	0.63	1.89
Starch	76.07	67.60	64.80	78.93	77.96	39.81
Soluble carbohy- drates	5.66	14.29	13.69	3.56	5.19	36.57
Albuminoids . . .	7.47	..	7.16	10.51	9.24	13.54
Gum, cellulose, etc.	undeterm'd	..	2.94	0.50	..	0.49
Ash	"	..	1.06	1.16	0.60	1.44

By heating, the albuminous substances also become considerably more soluble in water. Wheat flour, which in its original condition yields a very considerable amount of crude gluten on washing, after baking leaves a much smaller quantity. For the same reason a baked wheat flour may be mistaken

for barley flour, which has a non-glutinous dough. Along with these analyses may be given that of Robinson's Patent Barley, which is flour prepared from ground pearl barley, and "A B C" cereal milk, which is made from wheat and barley meal:

	Robinson's Patent Barley.	"A. B. C." Cereal Milk.
Water	10.10	9.33
Fat	0.97	1.01
Starch	77.76	58.42
Soluble carbohydrates	4.11	20.00
Albuminoids	5.13	11.08
Gum, cellulose, etc.	1.33	1.16
Ash	1.93	

Flour-ball.—Much has been written on the use of "flour-ball" prepared by long-continued boiling of superior wheat flour tied up tightly in a bag. A sample thus prepared by Dr. J. Lewis Smith and analyzed at his request afforded the following results. It was boiled for five days, fifteen hours a day, or seventy-five hours in all, the bag being taken out of the water over night. The original flour was white; the boiled flour, after thorough drying and pulverizing, of a light-yellow color. Its taste was remarkably flat and insipid, the long-continued boiling dissolving out the fat, some of the soluble albuminoids, and mineral matters. It is possible that very different results might have been obtained from a flour of different character and boiled for a much shorter interval (Dr. Eustace Smith recommends but ten hours):

	Original Flour.	Same Boiled.
Water	9.546	10.55
Fat	0.766	none.
Starch	71.924	72.362
Soluble carbohydrates	5.120	5.178
Albuminoids	11.280	10.520
Gum, cellulose, etc.	0.835	1.028
Ash	0.506	0.42

Liebig's Foods.—In the preparation of the flour by means of diastase (Liebig's foods) equal parts of wheat flour and barley malt, a certain amount of wheat bran (added, it is said, for the sake of the adherent phosphates and nitrogenous matter), together with 1 per cent. of potassium bicarbonate, are mixed with sufficient water to make a thin paste. The mixture is allowed to stand at ordinary temperatures for several hours, and then heated to 150° until the conversion of the starch into maltose and dextrin is completed. It is then strained and the residue pressed and exhausted with warm water. The extract is evaporated in vacuum-pans at as low a temperature as consistent with rapidity of working, and then dried with stirring at a higher temperature, so as to be brought into pulverulent porous lumps. The author's latest examinations of samples of foods belonging to this class are as follows:

	Mellin's Food.	Horlick's Food.	Savory and Moore's.
Water	12.37	9.70	8.34
Fat	0.18	0.34	0.40
Albuminoids	10.07	10.43	9.63
Soluble carbohydrates	68.18	76.83	44.83
Starch			36.36
Gum, cellulose, etc.	5.45	0.50	0.44
Ash	3.75	2.20	0.89

The starch is absent when the process is complete, and such was the case with some of the samples tested; in other samples a considerable portion remained.

The preceding foods are ordinarily employed with milk, the mixture being made at time of feeding. Still another class remains in which the dextrinized or malted flour has already been evaporated with milk, and which is prepared with the aid of water only. They are of very different composition, as will be seen from the following table:

	Nestle's.	Anglo-Swiss.	Gerber's.	American-Swiss.	Franco-Swiss.	Wells & Richardson's Lactated Food.	Loeflund's Cream Emulsion.	Malted Milk.
Water	5.00	6.50	6.78	5.68	4.43	7.76	24.32	2.18
Fat	4.25	4.91	2.21	5.81	3.70	1.64	15.32	5.30
Albuminoids	11.00	10.26	9.56	10.54	13.00	11.85	8.23	15.83
Soluble carbohydrates	40.91	46.43	44.76	45.35	46.09	39.00	49.43	66.99
Starch	36.86	29.48	35.00	30.00	30.86	36.43	Undet.	5.57
Cellulose, gum, etc.	0.28	0.40	0.48	0.41	0.50	0.71	"	
Ash	1.70	2.02	1.21	1.21	1.42	2.61	2.60	3.13

In the preparation of these foods the flour is first made into a dough and baked. The resulting biscuit is then finely ground and mixed with various amounts of condensed milk and dried by a slow heat at a moderate temperature. This leaves a mixture in which the starch has been partly changed into dextrose, maltose, and dextrin; the albuminoids of the flour have undergone the partial decomposition spoken of in the case of the farinaceous foods; the casein has been dried into separate particles, and the lactalbumin has been coagulated. On the addition of water the saccharine and a small portion of the albuminoids dissolve; the main portion of the albuminoids, the casein, and the starch, are left undissolved.

In the actual preparation of farinaceous, Liebig's, and milk foods for use in the feeding-bottle, the adjustment of the relative proportions should be such as to afford a ratio between the fats, albuminoids, and saccharine materials as nearly the same as that in human milk as possible. By making the cow's milk the principal article of the mixture, and basing the approximation on such a ratio as will render the albuminoids not very different in their gross amount from that in woman's milk, foods of the following character may be obtained. Of course the constituents other than the albuminoids differ widely in their gross amounts, and what has been said before in relation to their relative values in nutrition must here be borne in mind also. Selecting one food of each class, Column I. represents a mixture of 3 parts of thoroughly dextrinized flour, 47 parts of cow's milk, and 50 parts of water; Column II. the same relative amounts of Mellin's food, milk, and water; and Column III. a mixture of 1 part of Nestle's food and 6 of water:

	I.	II.	III.
Fat	1.91	1.86	0.71
Soluble carbohydrates	3.17	4.11	6.82
Starch	1.94	. . .	6.14
Albuminoids	2.27	1.89	1.83
Ash	0.36	0.43	0.28
Total solids	9.65	8.29	15.78
Water	90.35	91.71	84.22

MODIFIED MILK AND PERCENTAGE MILK-MIXTURES.

By THOMPSON S. WESTCOTT, M. D.,

PHILADELPHIA.

Modified Milk.—Modified milk is a term applied to the product of a recently introduced method which aims to effect a recombination of the fats, proteids, and lactose of cow's milk, so as to produce mixtures yielding any desired percentage of each of these three essential ingredients. While mother's milk is to be taken as the type of what such a mixture should be, it is possible by this synthetic process to vary the percentage of any or all of its three elements to meet any desired modification. The method originated with Dr. Thomas M. Rotch of Boston, and was perfected with the collaboration of Mr. G. E. Gordon, a dairyman of wide experience. The result of their labors has been the establishment of milk-laboratories, the first of which was opened in Boston in 1891; and since that time other laboratories have been started in several of the principal cities of the Eastern and Southern States, in Montreal, and, most recently, in London. Each laboratory is supplied exclusively by a dairy under its absolute control, situated within a short distance by rail, so that not more than three to six hours shall intervene between milking and delivery at the laboratory. By this means the laboratory has complete supervision of the handling of the milk and the control of its herd of cows. No cow is accepted until proven to be free from tuberculosis by the tuberculin test, and the health of each animal of the herd is carefully watched. Moreover, the feeding is carried out in a thoroughly scientific manner; no silage or pasture-feeding is allowed, and only measured quantities of wholesome fodder are given, for the purpose of maintaining a constant analysis of the milk. Upon this principle of feeding depends the uniformity of results, for it has been found that the daily variation from the standard analysis of 4 per cent. fat, 4.5 per cent. sugar, and 4 per cent. proteids is so small as to be practically unnoticeable in the laboratory modifications.

Not only is the health of the animals taken into consideration, but equal attention is paid to the employés of the farm and the laboratory, looking to personal cleanliness and the exclusion of any possibility of contamination from infectious disease. More than this, sterilization of all bottles, implements, or utensils likely to contaminate the milk is carried out as a routine procedure. In a word, every effort is made to secure a practical asepsis of handling by attention to all the details now carried out in modern aseptic surgery. The result of all these painstaking precautions is shown in the production of a relatively sterile milk yielding a definite percentage of its constituents.

Briefly stated, the materials from which modified milk is produced are—centrifugal cream of 16 per cent. fat-strength (rarely a 32 per cent. cream is required for certain prescriptions); separated milk, from which practically all fat has been removed by the centrifugation yielding the cream; a sugar-of-milk solution of 20 per cent. strength; and ordinary sterilized lime-water. By combining these ingredients in varying proportions and making up to the required total quantity with distilled water, almost any desired combination of percentages of fat, sugar, and proteids can be produced with great accuracy.

The method at present does not include a modification of the inorganic salts, nor does it attempt to vary the proportions of casein and lactalbumin, but treats the total proteids as a unit.

After the materials have been combined in the total quantity required for a day's feeding the mixture is divided up into as many portions as there are to be feedings; these are poured into sterilized nursing-bottles, which are then stopped with cotton plugs. If so ordered, these bottles are subjected in the sterilizing apparatus to any desired degree of heat for the purpose of pasteurizing or sterilizing; they are then packed in convenient baskets, and are ready for delivery. By this means the infant receives the proper quantity for a meal directly from a sterile bottle, without any chance of contamination, after leaving the laboratory, from exposure to air or from unclean vessels.

These laboratories are managed just like a reputable pharmacy, and refuse to prescribe over the counter. Blanks are furnished in prescription form, a copy of which, with a sample prescription, is as follows:

R _x	Per cent.	Remarks.
Fat	3	Number of feedings . . . 8
Milk-sugar	6	Amount at each feeding . . 5 oz.
Albuminoids	1 25	Infant's age 5 mos.
Mineral matter	—	Infant's weight 14 lbs.
Total solids	—	Alkalinity 5%
Water	—	Heat at 155° F.
	100 00	

Ordered for *Baby Doe,*
5090 Blank Avenue.

Date, <i>Jan. 1st, 1893</i>	Signature, <i>Dr. A. B. C.</i>
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For sake of illustration it may be stated that a mixture conforming to the above prescription will be made up of cream, $7\frac{1}{2}$ ounces; separated milk, $5\frac{3}{4}$ ounces; sugar solution, $9\frac{1}{4}$ ounces; lime-water, 2 ounces; and distilled water, $15\frac{1}{2}$ ounces. A 3-6-2 mixture would contain cream $7\frac{1}{2}$ ounces, separated milk $13\frac{1}{4}$ ounces, sugar solution $7\frac{3}{4}$ ounces, lime-water 2 ounces, and distilled water $9\frac{1}{2}$ ounces. It will thus be seen that for the same percentage of fat the quantity of cream remains constant for the same total quantity, and that as the proteid percentage rises the quantity of separated milk increases, the sugar solution undergoing a slight decrease because of the greater proportion of milk-sugar present from the larger quantity of separated milk.

The experience of a large number of physicians in feeding healthy infants on modified milk has enabled the Walker-Gordon laboratories to tabulate the average percentages and quantities of mixtures that have proven satisfactory for varying ages, as follows :

Theoretical Basis for Feeding a Healthy Infant.

	Age.	Gastric Capacity.	Prescription.		
			Per ct. Fat.	Per ct. Milk-sugar.	Per ct. Proteids.
Premature infant,		Drachms.	{		
		2-6		1.00	0.20
				1.00	0.50
Birth at term,	Hours.	Oz.		4.50	0.75
	24 to 36	1	—	5.00	—
1st	Week,	1		2.00	5.00
2d	"	1½		2.50	6.00
3d	"	2		3.00	6.00
4th to 6th	"	2½-3		3.50	6.50
6th to 8th	"	3-3½		3.50	6.50
8th to 16th	"	3½-4½		4.00	7.00
16th to 24th	"	4½-5¾		4.00	7.00
24th to 32d	"	5¾-7		4.00	7.00
32d to 36th	"	7		4.00	7.00
36th to 40th	"	7-8		4.00	6.50
40th to 44th	"	8-8½		4.00	5.00
44th to 48th	"	8½		4.00	4.50
48th to 52d	"	9		4.00	4.50

These figures, it must be remembered, are to be taken simply as averages, since the weight, as well as the age, of the child must be taken into account as a guide of its digestive capacity. Each infant's needs must be studied before a satisfactory modification may be secured. If anything, these averages are a little too high for any but infants in perfect health and with unimpaired digestion.

Laboratory modification has given most satisfactory results in almost all cases where artificial feeding was required, but more especially in cases of chronic gastric or intestinal catarrh, where proteids are digested with difficulty and variations in their proportions from day to day keep the digestion constantly disturbed. Such an infant may fail to digest a modification containing 1 per cent. of proteids, but will begin to thrive when this percentage has been reduced for a time to 0.75, 0.50, or even lower. In such cases the physician is enabled to accurately vary the dosage of any one or more of the ingredients of his mixture. The method offers a decided advance upon any method hitherto introduced for the feeding of infants with a substitute for mother's milk. It is at once scientific, accurate, and rational.

As a general rule, it may be stated that after a satisfactory formula has been found the strength of the food may be increased gradually, but as rapidly as the child's digestion will permit.

In reference to the changes in formula that may be required in any particular case after a prescribed mixture fails to exactly suit the conditions, it may be permitted to quote Holt's admirable summing up :

"If not gaining in weight, without special signs of indigestion, increase the proportion of all the ingredients; if habitual colic, diminish the proteids; for frequent vomiting soon after feeding, reduce the quantity; for the regurgitation of sour masses of food, reduce the fat, and sometimes also the proteids; for obstinate constipation, increase both fat and proteids."

As a corollary to this it may be added that, except in hot weather or in cases of pre-existing milk-infection, sterilization or even pasteurization is unnecessary, and that either of these processes may favor or directly cause constipation. Lime-water may also have the same effect. For a child with healthy digestion lime-water may often be omitted, at first tentatively, without any bad results.

Home Modifications.—It is readily understood that milk-laboratories are as yet inaccessible to a large number of physicians, and that the process is somewhat costly. Fortunately, it is quite possible to apply its principles to home modification, provided the mother have ordinary intelligence and will appreciate the importance of scrupulous cleanliness in all the necessary manipulations. Several methods have been suggested. Rotch (*Pediatrics*) uses gravity cream and under-milk, obtained by allowing a quart of good milk (averaging 4 per cent. fat, 4.50 per cent. sugar, and 4 per cent. proteids) to stand in a jar in ice-water for six hours, and siphoning off 24 ounces from the bottom, which leaves, according to his estimate, 8 ounces of a 10 per cent. cream in the jar. Holt, in his recent text-book (*Diseases of Infancy and Childhood*), proposes dilutions of various percentage creams with solutions of milk-sugar varying in strength from 5 to 10 per cent. According to this method, 16 per cent., 12 per cent., or 8 per cent. cream and whole milk (4 per cent. fat) are used with solutions of milk-sugar of 5, 6, 7, 8, and 10 per cent. strengths. An important fact to be remembered is that cream is practically a superfatted milk, essentially differing otherwise from milk in containing a slightly lower percentage of proteids, which vary from 3.20 for 20 per cent. cream to 3.90 for 8 per cent. cream, as contrasted with 4.00 in the average whole milk from which the creams are obtained; and that the sugar percentage is also slightly less than that of the milk.

Sixteen per cent. of butter-fat is about the strength of ordinary skimmed cream which has had about twelve hours to rise. It averages 3.60 proteids.

The 12 per cent. cream may be obtained by mixing two parts of 16 per cent. cream and one part of whole milk, or by skimming average milk after standing in a jar in iced water for about six hours. It averages 3.80 proteids.

Eight per cent. cream may be obtained by mixing one part of gravity cream and two parts of whole milk, or by skimming the milk after standing four to five hours. Removal of the lower milk by siphoning is less likely to disturb the cream layer, and thus partially dilute the cream. Eight per cent. cream averages 3.90 proteids.¹

These percentages are approximately correct, provided the whole milk maintains a fairly constant average value of 4 per cent. fat and 4 per cent. proteids. Variations here will of course disturb the cream percentages, but for ordinary cases the results are sufficiently close.

The sugar solutions may be made by dissolving an ounce of milk-sugar in 20 ounces, $16\frac{1}{2}$ ounces, $14\frac{1}{4}$ ounces, $12\frac{1}{2}$ ounces, or 10 ounces of boiled or distilled water to produce 5, 6, 7, 8, or 10 per cent. solutions respectively. The use of solutions of such varying strengths enables the modifications to be made without the use of additional plain water, and thus simplifies the preparation.

For comparison the following tables of dilutions of cream have been accurately worked out:

¹ The percentage figures used by Rotch and Holt, and also in the cream and whole-milk modification later described, are the standard analyses of the products of the Walker-Gordon dairies.

TABLE I.—*Sixteen Per cent. Cream.*

(Fat, 16.00; Sugar, 4.20; Proteids, 3.60.)

1 part of Cream to—

15 parts 5% Sugar solution =	Fat, 1.00;	Sugar, 4.95;	Proteids, 0.23
15 " 6 " =	" 1.00;	" 5.89;	" 0.23
15 " 7 " =	" 1.00;	" 6.82;	" 0.23
9 " 5 " =	" 1.60;	" 4.92;	" 0.36
9 " 6 " =	" 1.60;	" 5.82;	" 0.36
9 " 7 " =	" 1.60;	" 6.72;	" 0.36
7 " 5 " =	" 2.00;	" 4.90;	" 0.45
7 " 6 " =	" 2.00;	" 5.77;	" 0.45
7 " 7 " =	" 2.00;	" 6.65;	" 0.45
5.4 " 5 " =	" 2.50;	" 4.87;	" 0.56
5.4 " 6 " =	" 2.50;	" 5.72;	" 0.56
5.4 " 7 " =	" 2.50;	" 6.56;	" 0.56
4.3 " 5 " =	" 3.02;	" 4.85;	" 0.68
4.3 " 6 " =	" 3.02;	" 5.66;	" 0.68
4.3 " 7 " =	" 3.02;	" 6.47;	" 0.68
3.6 " 5 " =	" 3.48;	" 4.83;	" 0.78
3.6 " 6 " =	" 3.48;	" 5.61;	" 0.78
3.6 " 7 " =	" 3.48;	" 6.39;	" 0.78
3 " 5 " =	" 4.00;	" 4.80;	" 0.90
3 " 6 " =	" 4.00;	" 5.55;	" 0.90
3 " 7 " =	" 4.00;	" 6.30;	" 0.90
3 " 8 " =	" 4.00;	" 7.05;	" 0.90

TABLE II.—*Twelve Per cent. Cream.*

(Fat, 12.00; Sugar, 4.30; Proteids, 3.80.)

1 part of Cream to—

11 parts 5% Sugar solution =	Fat, 1.00;	Sugar, 4.94;	Proteids, 0.32
11 " 6 " =	" 1.00;	" 5.86;	" 0.32
11 " 7 " =	" 1.00;	" 6.77;	" 0.32
7 " 5-7 " =	" 1.50;	" 4.91-6.67;	" 0.48
5 " 5-7 " =	" 2.00;	" 4.88-6.55;	" 0.63
3.8 " 5-8 " =	" 2.50;	" 4.85-7.12;	" 0.79
3 " 5-8 " =	" 3.00;	" 4.82-7.07;	" 0.95
2.4 " 5-8 " =	" 3.53;	" 4.65-6.76;	" 1.12
2 " 5-8 " =	" 4.00;	" 4.77-6.77;	" 1.27

TABLE III.—*Eight Per cent. Cream.*

(Fat, 8.00; Sugar, 4.40; Proteids, 3.90.)

1 part of Cream to—

7 parts 5-7% Sugar solution =	Fat, 1.00;	Sugar, 4.92-6.67;	Proteids, 0.49
3 " 5-8 " =	" 2.00;	" 4.85-7.10;	" 0.97
1.6 " 5-8 " =	" 3.07;	" 4.77-6.62;	" 1.44
1 " 5-10 " =	" 4.00;	" 4.70-7.20;	" 1.95

TABLE IV.—*Four Per cent. Cream (whole milk).*

(Fat, 4.00; Sugar, 4.50; Proteids, 4.00.)

1 part of Milk to—

11 parts 5-7% Sugar solution =	Fat, 0.33;	Sugar, 4.96-6.79;	Proteids, 0.33
7 " 5-7 " =	" 0.50;	" 4.94-6.69;	" 0.50
3 " 5-8 " =	" 1.00;	" 4.87-7.12;	" 1.00
1 " 5-10 " =	" 2.00;	" 4.75-7.25;	" 2.00

3 parts of Milk to—

1 part 5-10% Sugar solution =	" 3.00;	" 4.62-5.87;	" 3.00
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It will be noticed that by these various dilutions of cream, and by intermediate dilutions not carried out in the tables, a large number of combinations of fat and sugar can be obtained, but that the proteid percentage in any instance must bear the same ratio to the fat percentage as holds in the cream from which the dilution is made. Low or mean percentages of fat

with high percentages of proteids cannot be obtained without additional proteids from skimmed milk. The practical value of the method therefore ends with a 1:1 dilution of whole milk. Finer variations in the relative proportions of fat and proteids, which are easily managed in laboratory modification, are thus impossible by the method of cream dilution.

Modifications with Cream and Milk.—For the reasons just stated, as well as the greater convenience in using whole milk as a basis of the mixture, and making up the necessary fat-value with additional cream, the writer has for some time been using such a method for home modification. It has been found that most satisfactory results can be obtained by using a 12 per cent. cream and whole milk (averaging fat 4.00, sugar 4.50, and proteids 4). It is first necessary to decide upon the number of ounces of total mixture, and fix the proteid and fat percentages desired. Then the number of ounces of mixed milk and cream can be found by the proportion

$$(1) \quad 3.90 : P :: Q : x,$$

in which Q is the total quantity of mixture, and P the proteid percentage: the value of x gives the number of ounces of milk and cream required to give the chosen percentage of proteids. The value of x being found, it remains to divide this quantity into two parts, C and M , the first of which represents the quantity of cream required, the second the quantity of milk. This is readily done by means of the formula

$$(2) \quad C = \frac{Q \times F - 4x}{8},$$

in which Q represents the total quantity of mixture, F the fat percentage desired, and x the quantity of mixed milk and cream already determined by formula (1). The quantity of milk, M , is at once found by subtracting the value of C from that of x . To illustrate: let it be desired to find the quantities of milk and cream to make a mixture of 40 ounces containing proteids 1.50 and fat 3.00.

Formula (1) becomes

$$3.90 : 1.50 :: 40 \text{ oz.} : x,$$

$$x = 15.4 \text{ oz.}$$

whence

Equation (2) becomes

$$C = \frac{120 - 61.6}{8} = 7.3 \text{ oz.}$$

and consequently, $M = 8.1$ oz.

Taking the same example, let it be required to get 4 per cent. of fat. The total quantity of milk and cream will be the same as in the previous case, but the quantity of each will differ. Here, from formula (2), $C = 12.3$ oz., and consequently $M = 3.1$ oz. The remainder of the 40 ounces of total mixture is to be made up by the addition of boiled water, barley-water, oatmeal-water, or whatever diluent is chosen. Lime-water, if desired, may also be added to the mixture in the proportion of 5 to 10 per cent. In the above examples 2 to 4 oz. of the diluent would be lime-water.

It will readily be seen that the calculation of proteids is not quite exact, since the varying proportions of milk and cream cause variations in the average value of the proteids; but, at the most, these vary only between

3.80 and 4.00, so that an average of 3.90 very satisfactorily represents this value. It is also evident that this assumed constant factor cannot be used for a proteid percentage higher than itself; but as such a combination would consist almost entirely of whole milk, the constant (3.90) should be taken very close to 4.00. For instance, if a 4.00 fat and 3.90 proteid mixture were desired, the constant factor should be taken as 3.99, and it would be found from formula (1) that 39.1 oz. of mixed milk and cream would be needed, the proportions of 0.4 oz. cream and 38.7 oz. milk being obtained from the other formula.

There are a few exceptions to the universal application of these formulæ that should be noted. In proteid values lower than 1.00, 16 or even 32 per cent. cream may be required;¹ in proteid values of 1.00 to 1.25, 16 per cent. cream is required for fat values from 3.25 to 4.00 for the lower, and from 3.75 to 4.00 for the higher of these proteid percentages; also, in the higher proteid percentages (2.25 to 4.00) skimmed milk, instead of cream, would be required for fat percentages lower than the proteid percentage. In practice, however, it is extremely rare to use a fat percentage lower than the proteid, so that this method of combination will be found to give most satisfactory working results, which come closer to accurate percentages than either cream-and-undermilk or diluted-cream mixtures.

The estimation of the quantity of sugar to be added for any desired percentage is considerably simplified by the fact that, since the quantity of mixed milk and cream remains constant for the same proteid value, the sugar to be added is also constant for the same sugar percentage: the variations in the fat percentage do not alter it. The quantity of dry sugar of milk to be added to the mixture to produce any desired percentage of sugar, S , is rapidly calculated from the formula

$$(3) \text{ Sugar} = \frac{Q \times S - 4.40 x}{100}.$$

In the examples already given, to obtain a 6 per cent. sugar mixture there must be added about $1\frac{3}{4}$ oz. of dry sugar.

A distinct advantage of this method is that if the quantity of cream be kept constant and the milk gradually increased, the total quantity of mixture being kept constant, both the proteid and fat percentages are gradually increased by an equal increment. When the fat value surpasses 4.00, beyond which it is rarely desirable to go, a half ounce may be dropped from the quantity of cream and its loss supplied by a half ounce of milk. From this point an increase of two or three ounces of milk may be made before the fat value again rises above the point desired, when another half ounce of cream may be replaced with milk. By this means the strength of food may be gradually increased without necessitating frequent changes of formula.

¹ When 16 or 32 per cent. cream is used, the denominator 8 in formula (2) should be made 12 or 28, and the constant factor in formula (1) should be changed to 3.80 or 3.45, to correspond. See papers on this method, *Archives of Pediatrics*, Jan., Feb., 1898.

SEA-AIR AND SEA-BATHING IN CONVALESCENCE.

BY W. M. POWELL, M. D.,
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THE difference between the air of an inland town and that of the sea-coast is that the latter is not only pure, but is saturated with sea-salts from the breaking of the waves upon the shore and the dashing of spray, which is carried toward the land by air-currents. If the wind is blowing from the sea, this characteristic saline odor may be noticed for some miles inland, but during a "land-breeze" it is hardly perceptible, even upon the beach. E. Freidick, in the *Southern California Practitioner*, quotes a large number of observers who have demonstrated the presence of sodium chloride in the air at the seaside, and shows that while there is naturally a small proportion of salt in this atmosphere, the greatest part of it is due to the diffusion of minute particles of sea-water. The proportion of salt is increased during strong winds, which blow the fine spray inland.

The air of the sea has a peculiar odor which is difficult to define, but which it is impossible to forget when once it has been inhaled. This odor, which is caused by the evaporation of the extractive matter contained in sea-water, is stronger when the waves dash upon rocks covered with sea-weed than when they break gently upon a sandy shore. It is also more perceptible during a storm than when the sea is calm.

Upon the border of the ocean the air is under greater pressure than in places of greater elevation, and consequently it contains more oxygen. The range of the barometer, the thermometer, and hygrometer is reduced to a minimum. These facts are only too often neglected in our estimates of the qualities of sea-air; they are, however, in a great measure responsible for the benefits derived by invalids during a residence at the sea-shore.

Ozone is one of the constituents of the atmosphere which is found in abundance on the sea and adjoining coast. Schönbein, its discoverer, believed it to be naturally formed out of atmospheric oxygen by the electrical discharges constantly taking place in the air. It is a most powerful oxidizing agent, so destructive to organic miasmata that its mere presence is a warrant of the absence of such noxious elements. It is more abundant by the sea than inland, and in windy than in calm weather. It is well known that the climate of any place where ozone is found in abundance must be healthy and exhilarating; hence we have at the sea-shore a pure air, containing oxygen in the form of ozone, besides finely divided sea-salts, as well as water which is rendered stimulating by the presence of the same salts. In most cases the breathing of this air has a marked invigorating effect, causing a great improvement in the appetite, promoting digestion and almost immediately producing a delightful exhilaration of the entire system. "No doubt can be entertained, in view of often-observed facts, that the effect of exposure to sunlight upon animal life is directly invigorating; and when with this is combined the constant inhalation of salt-air, and the daily application of salt water to the whole

surface of the body and limbs, it is easy to see why children should gain health and strength at the sea-shore."—*Packard*.

The temperature on or near the sea may certainly lay claim to greater uniformity than is obtained in localities remote from the coast. During the summer months the heated air of the land may be replaced by the cool breeze from the sea, while in winter the temperature of the coast-line is raised by the admixture of the warmer air from the sea with the colder air of the land. It is estimated that the Gulf Stream in this latitude during winter imparts to the air in contact with it a temperature of at least ten or fifteen degrees above that of the atmosphere of the earth, so that the ocean air in mixing with that of the land imparts to it an agreeable mildness which is unknown in the interior. Another favorable condition is found in the fact that the warmer air from the sea holds a large amount of invisible aqueous vapor in suspension, and as this commingles with the colder air of the land, it is condensed, gives out its latent heat, and becomes visible in the formation of clouds, especially at sundown. Thus that radiation of heat from the earth's surface into space which always takes place on clear nights is prevented. We can therefore safely assume that the mean temperature of the sea-coast is neither so high in the summer nor so low in the winter as that which prevails in the interior. These facts are well illustrated in the following table, prepared by Sergeant W. D. Blythe from the reports of the United States Signal Office, giving for five well-known localities the mean temperature for each month and the year, computed from November, 1879, to December, 1884, together with the average temperature for each of the four seasons:

	Winter.				Spring.				Summer.				Autumn.				Year.
	Dec.	Jan.	Feb.	Average.	Mar.	April.	May.	Average.	June.	July.	Aug.	Average.	Sept.	Oct.	Nov.	Average.	
Atlantic City, N. J. . . .	36.8	32.4	35.7	35.0	38.6	46.7	57.8	47.7	66.9	72.6	71.6	70.4	68.8	58.5	44.5	57.3	52.5
Barnegat, N. J.	36.4	31.9	35.1	34.5	38.3	46.0	57.2	43.8	65.5	72.2	71.1	69.9	68.0	57.7	44.2	56.6	52.0
Boston, Mass.	31.4	26.4	30.1	29.3	33.9	43.6	55.3	44.3	65.8	69.9	68.8	68.2	63.5	51.7	40.0	53.4	48.4
New York City.	34.4	30.0	33.6	32.7	36.7	47.0	59.3	47.7	68.3	72.6	71.6	70.8	67.5	56.2	43.2	55.6	51.6
Philadelphia, Pa. . . .	36.1	31.7	37.1	35.0	40.2	49.9	62.6	50.9	71.5	75.1	73.7	76.8	69.3	57.7	44.6	57.2	54.1

As a sea-breeze prevails on a large majority of the days during the summer months, the average summer temperature is much lower on the sea-coast than farther inland. On some days the difference is greatly marked, and few of us have failed to experience the relief afforded by the first breath of sea-air after spending a day in the hot city.

It is self-evident that the pleasantest climatic conditions are those which present the most even temperature, with only a moderate amount of wind and rain. The tables on the following page, compiled from the same source, give some interesting statistics of rainfall, temperature and wind at various well-known stations of the Signal Office.

Touching the question of health, the national mortuary table offers important data. There we find that while such model cities of the interior as Rochester and Milwaukee, swept as they are by the cleansing winds of the great lakes, show a death-rate respectively of 23.39 and 24.52 per 1000; while Philadelphia, the healthiest, save London, of the world's great cities, shows 21.20; and while nearly thirty people to the thousand die annually in Charleston—the death-rate among the resident population of a sea-coast town like Atlantic City is 12.5. There are only two places in the United States—Ashtabula, Ohio, and Los Angeles, California—where the death-rate shows any approximation to this last percentage.

Annual Precipitation, in inches and hundredths, as recorded at the U. S. Weather Bureau Stations on or near the Atlantic Coast, 1882 to 1891, inclusive; also the Average Annual Precipitations, computed from observations covering periods of from three to twenty-one years.

Stations.	1882.	1883.	1884.	1885.	1886.	1887.	1888.	1889.	1890.	1891.	Average amount.
Asbury Park, N. J.								57.56	55.03	51.64	3 yrs. 53.44
Atlantic City, N. J.	55.29	44.64	53.70	38.45	44.80	37.76	44.10	38.43	33.04	43.04	18 " 42.81
Baltimore, Md.	42.11	40.52	43.88	46.04	53.11	43.59	43.53	42.35	46.96	54.21	16 " 43.11
Barnegat (closed).											8 " 50.20
Block Island, R. I.	57.65	39.69	63.05	39.37	54.50	44.55	29.18	32.80	31.51	39.03	11 " 44.43
Cape May (closed).											10 " 46.70
Charleston, S. C.	57.01	51.35	60.22	67.93	65.94	44.61	49.46	52.25	47.84	45.90	16 " 58.92
Jacksonville, Fla.	53.26	53.34	53.92	52.04	54.86	58.60	53.13	46.22	47.52	41.32	15 " 51.04
Narragansett Pier, R. I.											5 " 52.38
New Orleans, La.	50.18	69.85	60.01	64.18	54.83	64.97	45.15	48.45	47.17	38.62	23 " 51.78
Newport (closed).											6 " 59.98
New York City	46.61	38.83	53.34	42.32	46.73	46.63	52.95	58.68	52.30	51.44	21 " 45.76
Norfolk, Va.	57.67	54.30	45.05	43.25	34.33	47.74	56.64	70.72	50.22	50.63	21 " 52.21
Portland, Me.	38.94	31.99	52.51	39.75	52.63	49.07	34.24	41.92	51.97	43.28	20 " 42.68
Sandy Hook	32.14	42.09	52.72	38.42	closed.						12 " 50.40
Washington, D. C.	46.79	45.71	49.96	44.84	58.17	45.38	61.33	41.59	52.95	51.22	21 " 45.06
Wilmington, N. C.	52.29	64.00	62.70	60.42	56.43	51.47	55.07	59.31	41.33	48.00	21 " 56.24

Monthly and Annual Mean Temperatures for 1889.

Stations.	Jan.	Feb.	Mar.	Apr.	May.	June.	July.	Aug.	Sept.	Oct.	Nov.	Dec.	Mean.
Asbury Park, N. J.	36.2	28.5	40.4	49.1	62.5	69.6	71.5	70.8	66.6	51.9	45.6	42.1	52.9
Atlantic City, N. J.	37.6	29.5	38.8	48.6	59.0	66.2	71.8	69.3	64.4	51.8	47.0	43.6	52.3
New York City	37.6	28.0	41.5	51.6	62.0	70.4	73.5	71.5	65.8	52.0	46.0	41.4	53.5

Annual Movement of Wind, in miles, at U. S. Weather Bureau Stations on the Atlantic Coast for ten years, ending Dec. 31, 1891.

Stations.	1882.	1883.	1884.	1885.	1886.	1887.	1888.	1889.	1890.	1891.	Average.
Atlantic City, N. J.	86.498	80.769	75.232	76.150	79.553	74.879	88.825	104.930	102.520	106.500	87.585
Barnegat, N. J.	117.564	128.939	125.081	124.061	closed						(4 years) 123.911
Block Island, R. I.	132.595	130.575	127.478	122.608	125.698	132.975	147.384	148.944			(8 years) 133.531
Cape May, N. J.	123.041	128.330	134.584	closed							(3 years) 128.653
Sandy Hook, N. J.	122.601	128.933	139.149	144.879	138.672	closed					(5 years) 134.847

Diseases benefited by Sea-air.—It is often asked, What diseased conditions are benefited by a sojourn at the seaside? and, What, if any, are acted upon unfavorably? Dr. A. W. Bell, author of *Climatology and Mineral Waters of the United States*, says that, considering the purity of the vapor and perfect solubility of the salt, it is difficult to conceive of any possible state of the human system under which the inhalation of such air would be detrimental. I fully agree with this author, and believe that sea-air is preferable to any other during a tedious convalescence. I know of no place where children improve more quickly than at the sea-shore. I have studied this subject closely since 1883, when I was resident physician at the Children's Sea-shore House at Atlantic City, New Jersey. Since that time I have been connected with the same institution, where upward of seven hundred children, both convalescents from various acute (non-contagious) diseases and those affected with chronic ailments and strumous manifestations, are admitted yearly during the summer months. No one without experience can realize the benefit obtained by these little sufferers, who remain at the Home for a fortnight to several weeks, according to the gravity of their cases. Here are sent, chiefly from Philadelphia, desperate cases of entero-colitis, patients almost completely prostrated by the heat, and other moribunds. Yet nearly all recover through the influence of the sea-air and

clean, healthful surroundings, with little or no aid from medicine. During the summer of 1892, in the latter part of July and the first week in August, the heat in Philadelphia and vicinity was intense. At this time I had more cases of severe entero-colitis than for several years, but they all recovered rapidly, save one, a child sixteen months old, who died four hours after its arrival at the coast. At the Children's Seashore House, where my friend Dr. W. H. Bennett was in charge, the cases were more severe than usual, but all terminated favorably.

It is an unusual circumstance for entero-colitis to develop at the sea-shore, and most of the cases seen there are brought from the neighboring cities or interior. Simple diarrhœa from indigestion, teething, etc. of course occurs.

Convalescents from scarlatina, measles, and the eruptive fevers generally do well by the sea.

The subacute nasal and pharyngeal catarrhs that we so often meet with in the spring as the results of repeated winter colds, which are usually so obstinate, invariably do well at the shore, where a complete cure is usually effected in a few days. Even cases of acute bronchitis seem to recover much more rapidly, and chronic forms are much improved. My experience with phthisis in children at the sea-shore has been limited: I have only seen a few cases, and they were far advanced. These children seemed to do well for the first week; the appetite improved, and sleep was more refreshing, although the cough remained about the same. After this they remained at a standstill, the improvement in appetite not being maintained and rest becoming disturbed again. These cases improved for the first few days when taken home, but fell back rapidly.

Asthmatic patients are frequently sent to the sea-shore, with, as a rule, most favorable results. Doubtless a long stay is beneficial to all such cases, especially those associated with chronic bronchitis. Patients arriving during a paroxysm nearly always experience an immediate relief, especially in cases of hay asthma; but should the attack originate at the sea-coast, removal to the city may in turn prove beneficial. Hyde Salter says: "I think it is a law, without an exception, that nervous affections are less prone to occur in proportion to the general bodily vigor, and what, for want of a more definite term, we must call the *tone* of the nervous system. Anything, therefore, that invigorates renders asthmatics less prone to their attacks. In this way sea-bathing is often of great service to asthmatics. By raising the standard of the general health it tends to prevent those humoral derangements which are often the exciting cause of asthma."

Cases of a strumous origin invariably do well by the sea: the appetite improves, the color returns to their cheeks, and they gain in flesh. Russel, who was the first to appreciate all the benefits derivable from the salt air, always had the hair of strumous children cut close, and exposed them freely to the cool sea-air with the neck uncovered; and he sent them back to their homes with their limbs strengthened and carrying in their countenances the evidence of the restorative powers of his remedy. When the strumous diathesis has further advanced, the effect of sea-air, although still of great utility, is much slower. There are many cases of cure, even when the glands of the neck have been greatly swollen, under the influence of two or three seasons passed by the sea. Roccas tells us that such a deeply ingrained constitutional disease as scrofula cannot be eradicated without a prolonged stay in a marine atmosphere. When the glands are ulcerated, Whitt many years ago recommended fomentations with sea-water and poultices made with it. It is supposed to facilitate the resolution of the swollen glands, even when they have become very

large and have existed for a long time. The following case, reported by Robert of Marseilles, fully confirms these assertions: "A lady coming from the interior of France brought to me her son, about fifteen years of age. The youth was enfeebled to the last degree, having been ill ever since he was nine years old. During all this time he had labored under scrofula of the neck, which was entirely surrounded with cicatrices of old ulcers. At the time I saw him the right elbow and one of the feet were affected; the elbow-joint was not diseased interiorly, but the ligaments which surrounded it were; and there were fistulous openings which had persisted for a length of time. As regards the foot, it was puffy and much enlarged, and he could scarcely bear it to be placed upon the ground: abscesses had formed several times, which had cicatrized, but there was another now threatening to open on one side of it. The most alarming feature of the case, however, was the terribly low state of the patient's constitution. His spirits were dejected; his face had the look of one prematurely old; his skin was dry and flabby; and his limbs almost entirely denuded of their flesh. Moreover, he was tormented with an almost continual diarrhœa. I advised the mother to establish her son upon the sea-coast, to make him pass the whole day upon the beach, and to make him use the sea-baths. Under this influence his general health began to improve, and then the swellings of the elbow and the thickening of the foot began to subside. Afterward I recommended that he should bathe daily, and that he should learn to swim. He fulfilled my orders so literally that he passed almost the whole of the latter part of the summer in the water. Always on the beach, he could find no other amusement so pleasant as that he derived from swimming. In a marvellously short time, considering the amount of disease, the youth was quite cured, and became what he still remains—a strong, healthy, and vigorous man."

Rickets is another common disease of childhood in which the benefits of residence by the sea are marked. The influence of sea-air upon this malady seems to exert a marvellous amount of good, and West, in recommendation of it, says that "even where marked deformity has already taken place amendment will be sure to follow." I fully agree with this authority, but will state that my experience in the past two years with this affection has been limited, as the stay of my patients during the summer months is hardly sufficient to show improvement if the disease is far advanced. But I do believe a prolonged stay by the sea, say a year or more, will bring about a complete cure.

Children suffering from Pott's disease, hip-joint disease, and arthritis of the knee all do well, gaining in flesh and improving in appetite without medical treatment.

Rheumatic cases, especially when chronic, do well by the sea-coast, and I know of no better treatment for this disease than warm sea-bathing. Fortunately for this class of patients, most prominent sea-coast resorts now can offer all facilities for warm sea-baths. These establishments are fitted with every convenience, including a lounging-room or "sun parlor," where one may take a nap after the bath. In cases of rheumatism the best results will be obtained from baths given on alternate days, followed by thorough friction of the body by a masseur or an intelligent nurse.

Cases of chorea during convalescence improve rapidly at the sea-shore. Although many writers highly recommend sea-bathing in this disease, I do not agree with them. Indeed, in one case, almost well, I am sure a relapse was occasioned by fright caused by a wave striking the child. Warm sea-baths, followed by a gentle massage, are preferable.

Sea-air has a very grateful influence in inducing sleep. Often sick children brought to the sea-coast sleep the first night better than for many nights before. It will be found that many children who are not ill after a few days' stay will complain of drowsiness and willingly take their afternoon nap.

The obstinate bronchitis which so often remains for an indefinite time after whooping cough is frequently cured by a few weeks' stay at the shore. In the paroxysmal stage of the disease, while the coughing spells are no less violent than elsewhere, children do not seem to lose flesh and color, no doubt because their appetite is kept up by the bracing effect of the clear atmosphere, and they are kept in the open air more than they would be in a city home.

Cases of infantile paralysis make a slow but steady improvement during a long stay by the sea. Most diseases of the skin and the inflammatory diseases of the eye are not improved by sea-air, unless these troubles have a strumous origin, in which case a long stay, by improving the general health, will indirectly improve the local condition.

Sea-bathing.—It is a popular belief that sea-bathing is both strengthening and hardening; and there is but little doubt that this opinion is well founded. It does not follow, however, that it should be practised by all without medical advice. Many hold that a plunge into water which is of lower temperature than air protects the system against attacks of catarrh and chill, and renders it indifferent to sudden climatic changes, whilst a few contend that perfect immunity from colds may be ensured by continuing the morning plunge throughout the year. We may say, without doubt, that sea-bathing, more than any other agent known, renders the body less sensitive to the influence of cold and to the injurious effects of prolonged exposure; but this, of course, is due to its invigorating and strengthening properties alone, and not to the element of temperature.

It is a remarkable fact that many persons who cannot profitably bathe in fresh water can do so in the sea; and the explanation doubtless is that the abstraction of caloric from the body in salt water is less than in fresh, by reason of its greater density. Probably, also, the saline ingredients have a more stimulating effect upon the skin and induce a more energetic reaction.

The most important characteristic of sea-water is its saline composition, and it is impossible to over-estimate the influence of the sea-salts in marine meteorology. It has been estimated that the average quantity of saline matter in sea-water is 3 per cent., consisting of chloride of sodium, sulphate of magnesium, sulphate of sodium, also muriate of magnesium and lime, with salts of iodine and bromine. Many, however, estimate the saline ingredients at 4 per cent. The above constituents are uniform as to presence, but are unequal in quantity in various parts of the world, so that in the Baltic a pint of water contains nearly forty grains of salt; on the coast of Great Britain it contains more than half an ounce; in the Mediterranean, much more; and in some ports south of the equator the quantity amounts to more than two ounces. It is in consequence of its saline character that sea-water does not evaporate from the skin so readily as fresh water. Even when the body is carefully dried particles of saline matter remain adherent, and find their way into the pores of the skin—as may be proved by the application of the tongue to the surface—and keep up a tingling glow long after the bath is over. We all know that persons when soaked to the skin by salt water do not take cold as easily as when caught in a shower of rain. This is explained by the fact that the pungent action of the sea-salts so stimulates the cutaneous circulation as to enable it to resist the depressing effects of the cold produced by the evaporation of the fluid portion. Sea-bathing, besides having all the beneficial effects of an ordi-

nary cold bath, has others peculiar to itself. The contact of the salt water and of the salt which adheres after the water left by the bath has evaporated stimulates the skin, increasing the circulation and exciting the sudoriferous glands. The beating of the waves against the surface of the body affords a passive exercise, with some of the advantages of massage; while to the more robust a healthful exhilaration and delightful active exercise are furnished by the plunge through the waves and the vigorous movements constantly required while in the surf.

At the resorts in the neighborhood of New York and Philadelphia the sea-bathing season is usually considered to be between the first day of June and the last day of September, as in this interval the temperature of the water ranges higher than at any other season.

The best time for taking a sea-bath is just before high tide. At that time the water has been somewhat warmed by passing over the hot sand. Moreover, the bathing is safer, from the fact that the tide still coming in would tend to wash the bather to the shore if he should lose his foothold, and, as the water covers a portion of the beach which was exposed to view a few hours before, there is less risk from dangerous holes and quicksands. But at most sea-shore resorts it has been found more convenient to bathe at the same hour each day—namely, at about 11 A. M., or two or three hours after breakfast, when the morning meal is digested and the system is beginning to feel the effects of the conversion of food into force, and is therefore better prepared to withstand the shock of the cold plunge. It is unwise, however, to bathe within two hours after any meal: whilst digestion is proceeding more blood is attracted to the digestive organs, in order that the process may be efficiently performed. But if we divert a portion of the blood to the surface of the body by the action of the cold bath, digestion is suddenly interrupted, assimilation checked, and congestive headache, cramps in the stomach, etc. are caused. In order to answer several of the questions which naturally arise, it is necessary to describe the phenomena, which are as follows: On entering the water there is a shock, accompanied by a sensation of chilliness and shivering; there is a respiratory embarrassment and a feeling of fulness in the head. Next follows a reaction, in which all these symptoms are relieved, and there is an agreeable sensation of warmth. If the bath is unduly prolonged, there follows another sensation of chilliness: the teeth chatter, the fingers and lips become blue, the respiration irregular and rapid, and the pulse weak and small. In the sea-bath each wave reproduces in a less degree the first shock, and at the same time hastens the development of the second chill. From the above description it would appear that the proper duration of the bath is a period short of the second chill, and the length of this period must depend upon the temperature of the water, the force of the waves, the strength of the patient, and a number of other circumstances.

I do not consider it wise to allow children to remain in the water over five minutes, and then they should be at once taken to their bath-house and not allowed to play on the beach in their wet bathing-suits. Before entering the water their heads should be wet, and they should be taken cautiously to the first line of breakers, where, in a stooping posture, the waves may wash over them. If children are afraid of the water, they should not be forced. The proper way is to accustom them gradually to the sea. Have them dressed in their bathing-clothes and allow them to play on the beach, when they will of their own accord go to the water's edge and gradually find their way in. Many children do not dread the water, and they may do much in allaying the fears of the more timid. I think three or four sea-baths a week quite sufficient

for even the strongest child. A thorough rubbing down should always be given, and the child quickly dressed, and allowed to resume its play in a sunny spot unexposed to the wind. There is no advantage in taking an infant (under two years) into the sea, and the practice as usually carried out seems almost inhuman; for these the heated salt-water bath is an excellent substitute.

The Management of Children at the Sea-shore.—At all times of the year the sea-shore is most beneficial to sick children, but it has only been a comparatively few years since the practice of going to the sea-side resorts during the winter and spring months came in vogue; previously, the three summer months were the only ones considered advisable to spend by the sea. At the present time it is deemed almost as necessary to take a child convalescing from an illness to the sea-shore in the winter and spring months as in summer.

In selecting a place of residence by the sea it is well to be near the surf. Houses situated at a distance from the beach are never as cool as those close to it. Therefore, in taking a sick child to the shore it is always advisable, especially during the summer months, to select a house in close proximity to the sea. Here the exhilarating breeze comes uncontaminated from the ocean.

The clothing of the child during its stay at the sea-shore should be slightly heavier than that worn in the city or country; hence it is always better to use woollen under-garments, light and loose in texture. Long stockings should invariably be worn, even in the warmest weather, as toward evening the air becomes several degrees cooler, and, if the breeze is blowing from the sea, at times almost cold.

Little change need be made in the food of children during their stay. The advantages, claimed by some authors, of a largely marine diet have probably been over-estimated, and much blame has been attached by others to fish, oysters, etc. for the frequent disorders of the digestive apparatus from which adults suffer at the sea-shore. From my own experience, however, the acute attacks of indigestion that we occasionally see are usually brought about by the elaborate menu which is found at our largest hotels, in contrast to the plainer home table which most are accustomed to. On arriving at the sea-shore the appetite is naturally sharpened by the change of air, and over-eating is the result.

Much thought should be given to the necessity of exercise. Children seldom need much urging, but the want of it among adults probably interferes with many of the benefits which otherwise would be gained.

For very young children, next to the walk in the nurse's arms, the drive upon the beach should be recommended. The perfect evenness of the surface renders it possible to take a very ill child into the open air frequently with the greatest benefit. One of the best forms of exercise for sick children is playing in the warm, dry sand. A spot should be selected where the sun does not beat too strongly, but which is at the same time perfectly dry. It is, as we all know, an unceasing source of amusement to children, and the harmless character of their little falls and tumbles during play often encourages them to efforts which they would not otherwise attempt.

PART I.

INJURIES INCIDENT TO BIRTH AND DISEASES OF THE NEW-BORN.

BY EDWARD P. DAVIS, A. M., M. D.,
PHILADELPHIA.

THE mortality of the first year of life is variously estimated. Bernheim, from an extensive series of statistics, places it at $37\frac{35}{100}$ per cent. of all children born. Winckel states that 10 per cent. of children born perish before the eleventh day of life; of these, $\frac{7}{10}$ per cent. perish during labor itself, $3\frac{3}{10}$ per cent. die as a consequence of some injury received during labor, while $2\frac{2}{10}$ per cent. perish from diseases contracted at or after birth. We shall first consider morbidity and mortality among children arising from injuries received at birth.

CAPUT SUCCEDANEUM.

The most frequent lesion sustained by the foetus during delivery is the formation of a tumor upon the head, usually known as caput succedaneum: this is commonly recognized after delivery as a somewhat boggy tumor, formed by infiltration of the scalp and fascia over the cranium, and usually situated upon the parietal bone opposite to that which came most in contact with the bony pelvis of the mother. The mechanism of its production is commonly thought to be as follows: In a normal presentation and position, the back of the child being to the left side of the mother's pelvis, and the vertex occupying the left anterior half of the pelvis, during the stage of expulsion the left half of the vertex of the child's skull receives the greater portion of the impact of force during descent and rotation. The continued pressure upon this portion of the foetal skull temporarily checks the free circulation of blood and lymph through the tissues of the scalp and fascia. There remains upon the opposite half of the vertex a portion of the head less pressed upon by the bony pelvis; here, naturally, the blood and lymph of the scalp-tissues are prevented from circulating through the left side of the foetal head by pressure, and accumulate and distend the tissues of the right half of the vertex. The result is a tumor upon the side of the foetal head opposite that which actually engaged during the first stage of labor. The position which the child's head occupied in the mother's pelvis may then be reasonably inferred from the location of the caput succedaneum; thus in the usual labor this tumor occurs in the right parietal region of the head. Should the child occupy a second position, its back to the right of the mother, its vertex situated in the right ante-

rior half of her pelvis, the caput succedaneum can be found upon the left parietal portion of the foetal head. Caput succedaneum is usually of no practical importance, as it disappears in a few days after labor. The infiltrated condition of its tissues, however, forms an excellent field for the growth of infecting bacteria. Should the mother's birth-canal be in a septic condition during labor, or should, through the carelessness of the nurse in washing the child, some injury occur to the tumor, the entrance of septic infection results in inflammation, and, in rare cases, in abscess of the scalp. The caput succedaneum is larger the longer the labor lasts, is usually of a bluish-red color, and does not distinctly fluctuate or pit upon pressure.

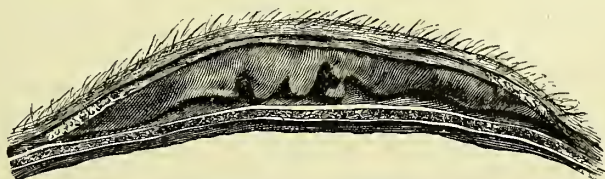
Occasionally the tumor embraces both parietal bones: this may be caused by long delay in the expulsion of the child, the head remaining for some time in the external genitals of the mother. Upon post-mortem examination extravasations of blood varying in size may be found in the vicinity of the tumor, and do not indicate criminal violence after birth. Two of these tumors may be found, a primary and secondary: the first is formed in the usual manner; the second is produced while the head is upon the pelvic floor and after anterior rotation has occurred. If delivery then be delayed, a secondary tumor will form, and may be distinguished from the first by its situation in the median line. In shoulder presentations the tumor is found upon the shoulder which presents.

So far as the treatment is concerned, Bouchut suggests the application of a solution of ammonium chloride, a solution of camphor, or an alcoholic mixture containing camphor. If this does not secure the disappearance of the tumor, he would aspirate it. Winckel and other obstetric authorities incise the tumor if it persists beyond the sixth or eighth day, and make pressure upon the parts with salicylated cotton. If abscess forms, incision and irrigation with a $\frac{1}{2}$ per cent. solution of creolin are indicated.

CEPHALHÆMATOMA.

By cephalhæmatoma Naegele, who first described it, designated a blood-tumor on the foetal head, called *true* cephalhæmatoma when beneath the periosteum of the skull, and *false* cephalhæmatoma when beneath the aponeurosis of the scalp. Virchow explains the formation of cephalhæmatoma by referring to the way in which the pericranium grows—namely, by proliferation of inner layers of the periosteum. If, then, the pericranium is separated from the cranium by the extravasation of blood, the bone-producing layers of the periosteum are still formed, but are prevented by the blood-clot from uniting with that portion of the bone for which they were intended. They join, however, to the bone at the border of the extravasated clot, where the bone is still attached.

FIG. 1.



Vertical Section through Cephalhæmatoma.

Much discussion has arisen as to the method of formation of cephalhæmatoma. Some ascribe its presence to traumatism only, while others seek an

explanation in a pre-existing condition of the infant's tissues. It is to be differentiated from caput succedaneum by several important distinctions. The latter arises during birth, is born with the child, appears upon that portion of the head turned during labor toward the excavation of the pelvis, is more prominent after difficult labors, has an ill-defined border, frequently crosses sutures, is discolored in appearance, and doughy upon manipulation, and tends to disappear rapidly after delivery. On the contrary, cephalhæmatoma does not occur, as a rule, after difficult labor, appears usually upon that parietal bone which did not present in the pelvic excavation, has a sharply-defined border, does not extend across sutures, does not discolor the scalp above the tumor, and usually gives the sensation of fluctuation in the centre of the mass. Cephalhæmatoma also tends to increase steadily in size for some time after labor.

With such radical differences the pathology of these tumors must differ widely. That of caput succedaneum has been already given. In studying the pathology of cephalhæmatoma we have been struck by the fact that instances under our observation have been, as a rule, in ill-nourished children born without especially difficult labor. In the wards of the Philadelphia Hospital we have frequently observed these cases in children born of ill-nourished mothers and poorly nourished at the time of birth. This leads us to believe that a pre-existing malnutrition lies at the basis of these tumors; thus, cases are reported where, in addition to the cephalhæmatoma, a profoundly anæmic condition of various organs of the child's body was present. In no case does this tumor occur as an extravasation of blood beneath the internal periosteum of the skull; but extravasations of blood within the cranial cavity are also described under the title of "intracranial cephalhæmatoma." Partridge describes two cases in which coagulated blood was found beneath the dura mater. No injury to the bones of the cranium existed in these cases, the brain-substance was softened, and the blood found beneath the membranes and at the base of the brain seemed to have been extravasated from the sinuses and from the laceration of minute blood-vessels. One of these children died very shortly after labor; the other survived for several days. We recall a similar case where delivery was easily effected by the forceps; the child perished, however, in thirty-six hours after birth, and upon post-mortem examination blood was found extravasated beneath the membranes, while the underlying cerebral matter was softened. Here also no injury to the bones, membranes, or sinuses could be detected.

Cephalhæmatoma is more frequently found in males than females, according to Burchard, in the proportion of more than three to one. The tumor is usually found upon the right side of the head. The children of primiparæ are most liable to this complication in the proportion of three to one. As a rule, cephalhæmatoma does not pulsate, although isolated cases are reported in which indistinct pulsation was observed. While fluctuation is usually present, it may be very obscure. This results from the presence of coagulated blood, as well as the breaking down of the clot in the centre of the tumor. It is observed that if the tumor be opened soon after formation, bright-red blood escapes; later the blood resembles the fluid found after old extravasation. The deposition of bony material on the under surface of the periosteum occasions a crackling sensation when the tumor is palpated. The fluid escapes irregularly from beneath the tumor; sensitiveness is very rarely a prominent feature. The bony ring surrounding the tumor forms gradually; thus Bouchut observed a case before birth in which no ring was present. Semmelweis is said to have seen cephalhæmatoma in a child delivered by Cæsarean section.

Several tumors may develop in the same individual; thus we recall a case under observation in the Philadelphia Hospital in which double cephalhæmatoma appeared on the head of a male child born after a normal labor. Triple cephalhæmatoma has been observed by Ovi after a precipitate birth in which

FIG. 2.



Double Cephalhæmatoma.

the infant fell to the ground, the cord rupturing three or four centimetres from the umbilicus. Upon examination a tumor was found upon each parietal bone, and one upon the occipital. The tumors were treated by incision and evacuation under careful antiseptic precautions, and uninterrupted recovery ensued.

The occurrence of cephalhæmatoma is readily understood when the loose attachment of the pericranium to the bone is remembered; Valleix found that in almost all infants ecchymosis between the pericranium and the skull is present after labor. It requires, then, but a constitutional liability to ecchymosis by reason of malnutrition to readily account for the occurrence of such tumors. Cephalhæmatoma, again, may develop after birth as a surgical injury, as instanced in cases described by Treves and Nélaton, as also in a remarkable case in a bleeder reported by St. Germain.

Cephalhæmatoma may be also produced by injudicious pressure exercised during the child's toilette. Hüter observed double cephalhæmatoma occurring on the fifth day after birth, and caused by the carelessness of a midwife, who, in washing the child, rubbed its head with undue force. The tumors persisted as long as the individual had charge of the child, but disappeared soon after she was discharged.

No one cause can be invariably assigned for the production of cephalhæmatoma: the size of the mother's pelvis seems to exercise but little effect, for Merttens in 21 cases found 6 in which the pelvis was normal, and only 5 in which slight pelvic contraction was present. In these cases the contraction was not of such nature as to interfere with labor. That the pressure of the pelvis has sometimes nothing to do with these cases is shown by Spiegelberg's observation of a case of premature birth at six months, in which the child perished before the rupture of the membranes; he was able to examine the head *in utero*, and detected the tumors before the expulsion of the child. He considered the tumors caused by interference with the oxygenation of the

foetal blood, and oftentimes by premature efforts at respiration. Merttens reports a similar case in which he diagnosticated this complication before delivery. The fœtus in this latter instance had a congenital hernia of the diaphragm, and hæmatomata were found in other portions of the body.

The diagnosis of cephalhæmatoma in distinction from caput succedaneum has already been stated. Hernia cerebri may be present, but occurs usually in the occipital region and in the line of sutures. Pressure upon the hernia produces symptoms of positive disturbance of the nervous system.

Aneurism presents a pulsating tumor of darker color, which neither has the appearance nor affords the history of cephalhæmatoma. The effort to class this affection among the hydrocephali is scarcely successful in the light of our present knowledge of both. Blood-tumors found in the occipital region in the dead fœtus are often caused by difficult labor, and are dark in color from the decomposition of effused blood. In encephalocele direct examination of the head by palpation will enable the physician to make the diagnosis. Tumors in living children, the result of direct violence, are usually painful on pressure and lack the sharp outline of cephalhæmatoma. Occasionally, in advanced rachitis, where craniotabes is present, soft pieces of bone in the skull may simulate a blood-tumor when palpation is made through the scalp.

The usual plan of treatment consists in making gentle pressure by a pad of antiseptic cotton conveniently held in place by a night-cap. Occasionally lotions containing dilute alcohol or some acetous preparation are employed, but there is no evidence of their positive value. It must be remembered that the tumor, as a rule, will have reached its largest size six or eight days after the birth of the child. Unless hæmorrhage be excessive and the tumor becomes rapidly very large, it may be let alone for the first ten days of the child's life. Should infection occur and inflammation supervene, it must be freely opened at once, emptied of its contents, and the sac thoroughly disinfected, while continuous but gentle pressure is made by an antiseptic dressing. If no complication occurs, at the end of the first eight or ten days of the child's life the scalp over the tumor should be shaved, the surface thoroughly disinfected, preferably with boric acid, and the tumor punctured with a bistoury or large trocar. After evacuating the fluid contents pressure by an antiseptic dressing is indicated. Some prefer free incision in place of simple puncture. We have met with a case in which puncture and evacuation were followed by reaccumulation of fluid, and in which it was finally necessary to open the tumor freely, empty it, and pack it with iodoform gauze, the gauze having to be renewed several times before adhesion between the bleeding surfaces took place. Occasionally the loss of blood in these cases is considerable; as a rule, however, hæmorrhage is not a serious complication.

The susceptibility of infants to poisoning by antiseptics should be remembered in treating cephalhæmatoma. Mercurial and carbolic solutions may be preferably replaced by solutions of thymol, 1:1000, or saturated solutions of boric acid. Iodoform gauze may be employed without hesitation as tampon material.

HÆMATOMA OF THE STERNO-CLEIDO-MASTOID MUSCLE.

A peculiar induration is frequently observed in the sterno-mastoid muscle of new-born children, regarding which different beliefs have been held. Anatomical study of the subject shows that the lesion is an intramuscular fibrosis, caused by direct violence to the neck of the child, usually occurring at delivery. Most of these cases result from delivery in breech presentation; the

forceps causes some; and, rarely, the lesion follows spontaneous birth. Schmidt reports the case of a child, seven days old, delivered by the breech, in which the right sterno-mastoid was shortened, and the right half of the face smaller and flatter than the left. The report of a post-mortem examination upon a case pointing to a possible intra-uterine origin of this condition is made by Heusinger. The head was directed toward the left, the right sterno-mastoid muscle was 9 cm. long, the left only $6\frac{1}{2}$, and was a soft, white, tendinous substance. In 23,293 children examined at birth at the Paris Maternité, Guyon found 132 cases of monstrosity, but no case of torticollis, which militates against the congenital occurrence of hæmatoma of the sterno-mastoid. In 64 post-mortem examinations Ruge found 13 cases of this complication. In a recent valuable paper Spencer describes 15 cases found in 300 autopsies; his researches show that both sexes and the muscles of both sides of the neck are equally affected. Small, prematurely-born children are especially liable to this injury. Breech or footling presentation was observed in 10 of the 15. The forceps had been employed in 2 cases, while in 2 no instrumental aid was employed: in 2 of the bodies examined both muscles were affected. Spencer notes but two cases of contracted pelvis; one of his cases was that of triplets, complicated by placenta prævia centralis, with extraction through perforation in the placenta. His microscopic sections show clearly rupture of muscular fibre, with extensive effusion of blood. It has been shown by Witzel that, as a consequence of this complication, contracting fibrous bands may form, giving rise to permanent wry neck. Jacobi believes that the forceps is frequently the effective agent in producing this injury to the fœtus.

HÆMORRHAGE IN THE NEW-BORN.

A considerable number of cases of foetal death occurring within the first forty-eight hours after labor are preceded by obscure symptoms which render an exact diagnosis difficult or impossible. The intelligent study of such cases by post-mortem examinations shows us that hæmorrhage is usually the cause of the fatal issue. As in the adult, hæmorrhage may depend upon an alteration in the condition of the blood itself, and also upon direct mechanical injuries which result in its escape from the vessels. In the first category of cases it has long been a familiar observation that syphilitic children, stillborn, show extensive disintegration of blood, with extravasation of blood-serum from the serous surfaces of the body. Children dying from acute infections on the part of the mother, and stillborn or perishing soon after, often display such a tendency to hæmorrhage; thus, small-pox, typhus, typhoid, scarlatina, and, as a rule, the acute infections as a class, predispose to the occurrence of hæmorrhage. There is also direct proof from bacteriological examination that the fœtus *in utero* may be infected by various micrococci, and that this infection may result in hæmorrhage and death at labor or very soon afterward. The occurrence of multiple punctate hæmorrhages accompanying umbilical sepsis is a not infrequent illustration of this form of hæmorrhage. In the recent literature of the subject Tavel and Quervian report a case of multiple hæmorrhage following umbilical infection by streptococci. Death occurred on the thirteenth day, the infection having occurred very shortly after birth. A thorough examination of the specimens showed infection with streptococci and other bacteria to be the cause of the hæmorrhages. These hæmorrhages were found in the connective tissue beneath the epidermis, beneath the serous membranes and mucous membranes, and also in the kidneys. A second illustrative case is also reported, in which, in a prematurely-born child, death occurred with symptoms

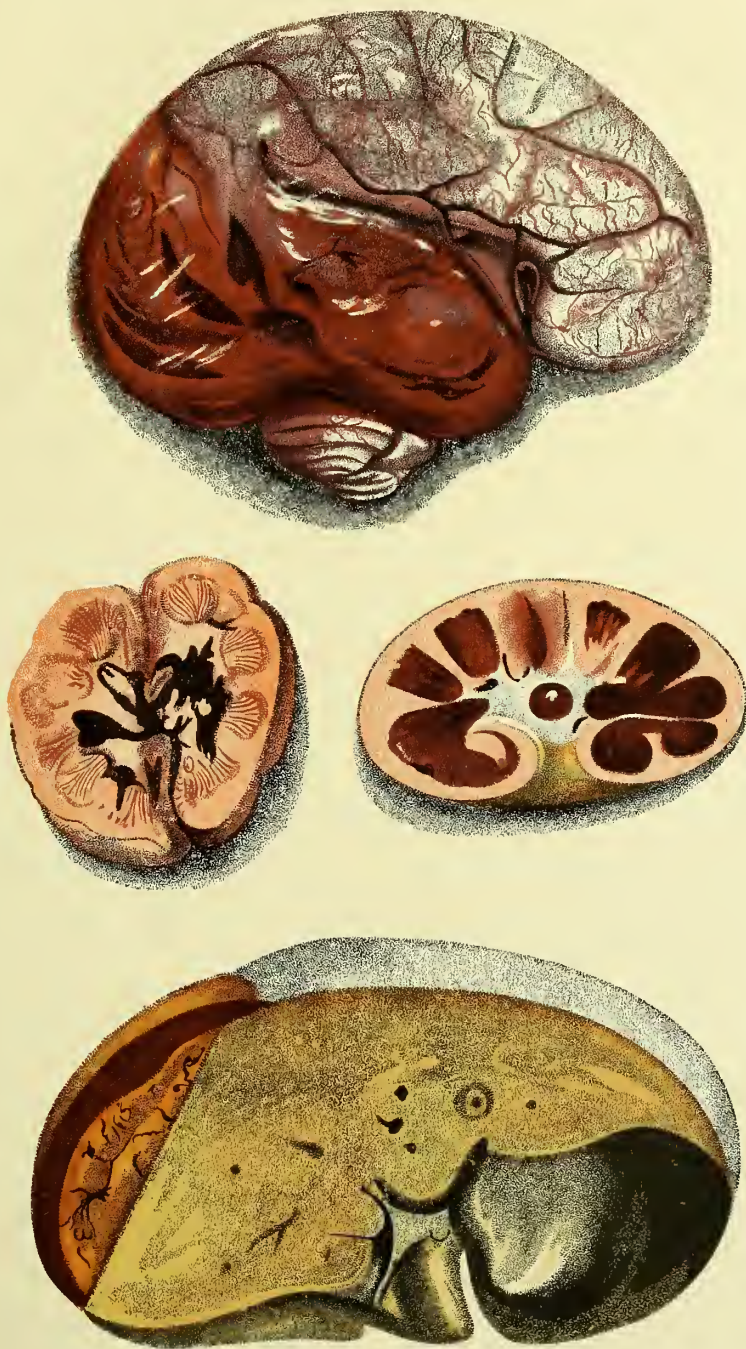
of pneumonia. Examination revealed the fact that the pneumonia had been caused by infection with staphylococci. The peculiar form of the hæmorrhage—namely, into the parenchyma of various organs—excluded hæmorrhage from mechanical injury. Further, the rapid and easy birth of a small foetus tended to exclude the possibility of mechanical injury.

By far the most frequent cause, however, of hæmorrhage in the new-born is direct mechanical injury received during birth. Such injury is usually suspected after difficult extraction by the forceps or by version. As pelvimetry is more extensively practised the induction of premature labor will render these cases more and more infrequent; but at present they occur with sufficient frequency to form an important complication of labor. Under the head of *apoplexy neonatorum* Ashby and Wright describe cases of hæmorrhage from the *pia mater* following compression of the umbilical cord and pressure upon the brain-substance during birth. Convulsions may be present in such cases, and if paralysis occurs it is probably peripheral, resulting from effusion of blood at the base of the brain, on the pons, or the origins of the cranial nerves. McNutt has reported 10 cases of cerebral hæmorrhage following labor; in 7 of these cases the head presented; in 3, the breech. In all the latter cases paralysis occurred, but only localized convulsions. McNutt infers that hæmorrhage, limited to the convexity of the cerebral hemispheres, is more apt to follow delivery in breech presentation.

Various forms of cerebral hæmorrhage are described by other observers, and especially in cases following prolonged application of the forceps or forcible extraction after version. In our own observation we recall the case of an infant delivered with axis-traction forceps without especial difficulty; progressive feebleness of respiration, failure to nurse, and apparent exhaustion caused death in thirty-six hours after birth. On post-mortem examination, over the parietal regions of the skull the tissues of the scalp were intensely congested, although no gross lesion, as rupture or fracture, could be discerned. Beneath these portions of the skull and scalp the cortex of the cerebrum was filled with punctate hæmorrhages, and over the point of greatest convexity the brain-substance was materially softened. Similar cases, which would not be found infrequent if post-mortem examinations in such patients were extensively held, are readily explained by the anatomy of the cranium and its contents in the new-born. Virchow and others have shown that the blood-vessels of the infant's brain are thin and small, and most readily injured by abnormal pressure. An interesting example of this fragility is found in cerebral hæmorrhage following death from asphyxia, where mechanical injury to the cranium can be excluded.

In medico-legal practice Richardière emphasizes the fact that such cerebral hæmorrhage may be differentiated from hæmorrhage occurring later in life by the absence of inflammation of the arachnoid and of the dura mater. Meningeal hæmorrhage in the new-born is often accompanied by subpleural ecchymoses; death usually results suddenly. A most valuable recent contribution to the literature of this subject is that of Spencer. In a total of 180 bodies examined, 130 were in a condition which enabled a critical examination of the tissues to be made: in 85 injuries to the brain were found, consisting of congestion and hæmorrhage; these conditions varied in severity, in situation, and in extent. Oedema was a frequent accompaniment. The children had been delivered in various ways, and many of the cases occurred in children the subjects of disease. The accompanying plate shows a typical condition of meningeal hæmorrhage (Plate I.). Its frequency will be appreciated when it is known that $4\frac{7}{10}$ per cent. of all hæmorrhages occurring in the new-

PLATE I.



Visceral Hæmorrhage in the Newborn (Spencer, *Transactions Obstetrical Society, London*, vol. 33).

born are meningeal in character. Spencer also describes a case, similar to the one which we have mentioned, of hæmorrhage into the substance of the brain. It is interesting to note that, so far as the causation of cerebral hæmorrhages is concerned, the forceps is the most frequent agent in producing them, and next presentation by the breech or foot. As determining causes softness of the skull and relaxation of the sutures are of considerable importance.

In Spencer's cases, next in frequency and importance to hæmorrhage into the brain comes parenchymatous hæmorrhage into the liver, kidneys, and suprarenal capsules. Well-marked congestion was frequently observed; hæmorrhage was present in $28\frac{46}{100}$ per cent.. This hæmorrhage was often upon the upper surface of the liver and followed birth in head presentations. Such hæmorrhages usually appeared as blebs filled with blood. Of equal frequency was hæmorrhage into the substance of the kidneys, usually beneath the capsule. Such cases were most frequent in breech presentations (Plate I.). The suprarenal capsules were also the seat of frequent hæmorrhage. Injuries to the lungs in the form of congestion and hæmorrhage were next in frequency. Most often this took the form of subpleural bleeding; less frequently, as hæmorrhage into the lung-substance.

These pulmonary apoplexies are often followed by pneumonia, and are a frequent cause of death. Such infants are usually cold and blue, with sub-normal temperature and feeble cry, and do not nurse. The abdominal and pelvic viscera, besides those mentioned, are also the frequent site of congestion and hæmorrhage. As regards the causes, Spencer recognizes a delicate condition of the blood-vessels as of great importance. Alteration of the blood, already described, is also recognized, while asphyxia predisposes to hæmorrhage. Direct mechanical violence is a familiar exciting agent.

Experience abundantly proves that most cases of severe hæmorrhage arise where disproportion in size between the fœtus and the pelvis exists; there can be no rational prophylaxis of these injuries that does not rest upon an estimate of the mother's size and the relative size of the fœtus. We cannot too strongly urge, as we have already done, that pelvimetry be uniformly practised by obstetricians, and that, in addition, an effort be made in all cases to estimate the relative size of the fœtus and the birth-canal. To be of service to the patient such efforts at diagnosis should be made between the seventh and eighth months of pregnancy.

So far as the treatment of the infectious disorders which attack the blood, resulting in hæmorrhage, is concerned, the faithful practice of antiseptic precautions will diminish very largely these complications. The need for such observances is proven by the familiar fact that at the present time the mortality of infants in private houses is greater than in well-conducted maternities, the reason being that the practitioner considers the private house and the private patient objects for less anxiety than the hospital patient; neglecting antiseptic precautions because the patient is a private one the result is often disastrous.

ASPHYXIA.

Interference with the oxygenation of the fœtal blood results in asphyxia. By far the most common and dangerous causes of this complication are those which arise while the child is still in the uterus, and which have nothing to do with the access of the external atmosphere to the child's lungs. When this is kept in mind, it will be seen that asphyxia is a complication of labor itself, not so much a condition arising at delivery and requiring subse-

quent treatment. The most frequent cause of this condition is pressure upon vessels of the placenta or umbilical cord, resulting in blood-stasis in the foetus; or occasionally sudden collapse and death on the part of the mother. The symptoms of asphyxia in the foetus are those of carbon-dioxide poisoning—a rapid, feeble pulse, pallid appearance of the surface of the body, with the phenomena caused by intense congestion of various organs, ending in heart-failure. Asphyxia has been variously divided, some writers describing an apoplectic form and others a pallid form. These are but variations of the same condition, and are distinctions without essential differences. During the first stages of asphyxia the phenomena of congestion predominate: the face of the child is suffused, the mucous membranes bluish, the heart-beat at first slow and more vigorous than normal, while the reflexes still remain. As the process goes on and congestion has been followed by engorgement and oedema, the surface of the body is pale, the pulse small, rapid, and feeble, while the mucous membranes have the peculiar grayish-blue appearance characteristic of impending death. In the first stages of asphyxia the pulse in the umbilical cord is present, and may be vigorous. In the second stage the cord is pulseless, and shares the pallid appearance of the foetus.

The complications of labor which most frequently cause asphyxia are partial detachment of the placenta, compression of the umbilical cord, pressure upon any large portion of the foetal body, especially upon the head and brain, or the sudden death of the mother. So soon as the tissues experience what has been styled “hunger for oxygen,” there ensue reflex respiratory movements: by experiment these may be demonstrated to happen within the uterus before the rupture of the membranes; they frequently occur during the second stage of complicated labor. They result in the inspiration of amniotic liquid or the secretions of the mother’s birth-canal; if these respiratory efforts are vigorous and prolonged, inspiration pneumonia may result—a catarrhal pneumonia caused by the inspiration of mucus or pus, developing, if the child survives, immediately after birth, and frequently proving fatal.

The child before labor is in a condition of physiological apnoea. The blood of the foetus contains an excess of hæmoglobin at the moment of birth, stated by Cattaneo to be relatively $120\frac{2}{10}$ per cent. No differences can be distinguished between arterial and venous blood in the umbilical cord in the amount of hæmoglobin contained. So perfect is the provision of nature for supplying the foetus with oxygen that anæmia on the part of the mother does not seem to influence the amount of hæmoglobin in the foetal blood nor in the blood of the child immediately after birth. The rapidity and ease with which the foetal blood absorbs oxygen is illustrated by the fact that in from thirty-six to forty-eight hours after birth the blood of the new-born contains its greatest amount of hæmoglobin. Late ligation of the umbilical cord results in more hæmoglobin in the foetal blood. Curiously enough, a small placenta increases the amount of hæmoglobin in the foetal blood, while a large placenta diminishes it. At the moment of birth the circulation of blood in the placenta and the child is markedly interrupted, oxygenation is materially lessened, and the foetus undergoes a period of more or less danger. It can be readily understood how delayed labor, where the exhausted uterus in tetanic contraction presses upon the child and the placenta, may occasion death from asphyxia, and this without extensive gross lesions.

Asphyxia, again, may depend upon defective muscular and nervous development in the foetus. As a result, the foetus fails to make respiratory movements after delivery, and perishes from actual weakness. Diseases which affect the respiratory apparatus, either by structural changes or mechanical pressure, may

cause asphyxia. Pulmonary syphilis, enlargement of the liver, dropsy, and various tumors come under this head. These cases usually perish from atelectasis. The blood-vessels in such cases rupture easily, and small multiple hæmorrhages abound.

Prognosis in cases of asphyxia depends upon the condition of the nervous centres. If the asphyxia is but partial, and the stage of congestion be present, as evidenced by the dark reddish-purple complexion of the child and the slow but full pulsations of the heart and umbilical cord, recovery in the majority of cases will ensue. If, however, the child is pallid, the heart-beat rapid and feeble, and the cord pulseless, the prognosis is grave. More than 1 per cent. of children born living perish from asphyxia; while cases have been reported where children, born asphyxiated, subsequently developed serious pathological conditions of the nervous system. Recalling what has been stated regarding the richness of the foetal blood in hæmoglobin, cases where children born asphyxiated have survived for hours, although thought to be dead, are readily explained. Beale described a case in which the mother died from post-partum hæmorrhage shortly after delivery; the midwife in charge reported the birth of dead twins, which she put in a basket in a shed; on examination three hours afterward, one child was found breathing feebly. Efforts to establish respiration were fruitless. The temperature in the shed was very low, the weather being cold. Children have respired feebly eighteen minutes after birth and twenty-five minutes after birth in breech presentation. Beale reports successful efforts, lasting several hours, to resuscitate a child thought to be dead. A case is reported where a child was buried a foot under ground, and not exhumed for five hours, when evidences of life resulted from efforts at resuscitation continued for two hours. It is curious to observe that the chances of recovery in asphyxia are much better when the infant is exposed to cold than when to heat, probably from the fact that a low temperature retards the metabolism of the cell-elements of the body, and thus the nervous centres retain their irritability longer.

Treatment of asphyxia is prophylactic and curative. In prophylaxis the conditions which will result in prolonged labor should be anticipated and removed. Complicating factors which will subject the child to great pressure must also be obviated. The judicious use of the forceps is a direct prophylaxis against asphyxia, as are version and extraction. On the other hand, both of these procedures are direct causes of asphyxia in unsuitable cases. We must again repeat that no intelligent prophylaxis of asphyxia can be undertaken which does not include a preliminary examination of the mother's birth-canal and an estimation of the relative size of the foetus and the mother. Prolapse of the umbilical cord, resulting in pressure and asphyxia, is best treated by anæsthetizing the mother and terminating labor, if possible, by manual interference; thus, the cord may be taken in the hand and passed up into the uterus, the head brought into a proper position, and delivery expedited by the forceps; or, if pulsation in the cord has ceased, version and extraction may be performed. There is no repositor for the cord comparable to the hand of the obstetrician, for the hand can recognize pulsation, can remedy coiling of the cord about the foetus, and may so change the position of the cord as to lead to the recovery of the foetus.

In cases of contracted pelvis, or in disproportion between the foetus and the pelvis, operative procedures have for one of their purposes the saving of the child from asphyxia, which otherwise must prove fatal. So soon as the head is accessible during labor, the practitioner should ascertain, if possible, whether the cord is coiled about the neck; if so, it should be gently drawn

down and loosened; and if the head be born, the cord tightly coiled about the neck, and a large body and shoulders hinder delivery, it is well to cut the cord and deliver the child rapidly. The cord may be clamped with artery-forceps, or, better, tied. The diagnosis of cord around the child may sometimes be made before expulsion by hearing a murmur in the umbilical cord during auscultation of the abdomen.

The treatment of the actual condition of asphyxia after delivery will depend largely upon the degree of asphyxia present. There are certain precautions which should be taken in every birth. The nurse should have ready a saturated solution of boracic acid to which has been added a teaspoonful of glycerin to the half pint. This should be at hand in a small, clean earthen bowl. In the bowl should be a half-dozen pieces of old, soft handkerchief, two inches square. When the head is born, the physician turns the mouth and eyes of the child in such a position that they will not come in contact with the discharges of the mother. The nurse or physician should then thoroughly cleanse the mouth and fauces with the bits of linen soaked in the boracic solution. Mucus or secretions in the child's mouth will thus be removed, and one danger of asphyxia obviated. In the stage of asphyxia where congestion is the principal symptom, the stimulus of contact with the external air will often secure respiratory movements: spanking the child is a familiar method of procedure which undoubtedly has good results. In such cases the cord may be promptly tied and cut; and if the congestion be pronounced, it is well to allow a drachm or two of blood to flow from the foetal cord before ligation. The child should then be promptly inverted to favor the expulsion of mucus from the air-passages. If the heart-beat be good, a little cold water sprinkled upon the chest will usually result in the establishment of respiration. Should the heart-beat be good, but respiration not ensue, the child may be laid in a bathtub filled with water at a temperature of 100° F., and passive respiratory movements may be instituted. Cold water also may be sprinkled upon the chest. In these cases a prognosis may be based upon the action of the heart; if that be strong, the physician should not despair of securing respiratory movements.

In the more severe forms of asphyxia the child can endure no loss of blood; it may be promptly inverted and held in that position for several moments, its mouth being thoroughly emptied of mucus and secretions: passive respiration is then to be instituted, and to secure the actual entrance of air into the lungs the Schultze method is undoubtedly pre-eminent. It consists in taking the child with both hands, the child's head raised between the upper portion of the palms, the fingers grasping the scapulæ of the child, the thumbs resting upon the anterior surface of the thorax. The child is then raised above the head of the physician until it turns a three-quarter somersault; it is then brought down with a swinging motion to within a short distance of the floor. When the body of the child is raised over the head of the physician expiration results: as the child swings forward and downward the action of gravity and the pressure of the physician's hands result in a powerful inspiratory action. The value of the Schultze method consists in its efficiency in introducing air into the lungs; it is not, however, a stimulus to the reflex excitability of the nervous system, and if this has been lost, an infant's lungs may be filled with air and yet the child readily perish. The dangers of this method have been pointed out by Meyer and Heydrich. Fracture of the clavicle with perforation of the lung and emphysema are reported by these observers as occasionally following this method of resuscitation.

A manifest objection to the Schultze method is the disturbance and shock

which must necessarily follow; in deeply asphyxiated children, where the heart-beat is scarcely perceptible, it is preferable to practise the inverted posture, with the application of warm flannel to the surface of the body and the continuation of gentle respiratory movements. Air may be introduced into the lungs by mouth-to-mouth insufflation or by the passage of a tracheal tube. Lusk advises the use of the catheter, not only to remove mucus, but to favor direct insufflation; or the chest-walls may be compressed to secure expiration. When circulation reappears, Silvester's method is then of service, the tongue of the child being drawn forward. When heart-beats are perceptible, the warm bath, with sprinklings of cold water upon the face, is useful. Finally, he advises Schultze's method to favor complete re-establishment of the circulation. Schultze claims for his method an immediate action in relieving overloaded blood-vessels, the swinging of the child producing emptying of the ventricles and favoring the return current from the pulmonary vein.

The value of direct insufflation by the catheter, preceded by the removal of mucus, can scarcely be over-estimated. We recall a case in a foreign hospital where the assistant in charge had abandoned an asphyxiated infant as dead; permission was given several American students to practise the passage of the balloon catheter, an English catheter having a rubber bulb at the distal end, whose compression and expansion favor suction and insufflation. To our surprise, the child became resuscitated under the use of the catheter, and ultimately recovered. Forest places the child first on its face, its head down, and expels fluids from the mouth by pressure upon the back. The child is then put in a bath or tub of hot water in a sitting posture, supported by one of the operator's hands across its back, its head bent backward. The physician grasps the child's hands with his other hand, carries them upward until the child is suspended by the arms, leans forward himself and blows air into the child's mouth; the infant's arms are then lowered, its body is doubled forward, and its thorax pressed between the hands of the physician. Air is thus expelled. Especial advantage is claimed for this method from the fact that the hot water maintains capillary circulation and tends to assist in promoting the action of the heart. Reynolds places the infant upon its back, head downward, resting upon the operator's forearm, held nearly perpendicularly to the floor, retained in that position by his fingers hooked over its shoulders. In this position the child's arms fall downward by the sides of its head, and their weight, aided by that of the thorax itself, draws the ribs into the position of complete expansion of the chest. The thorax is compressed against the forearm by the other hand, and suddenly released, when a most satisfactory respiration is the result. This method combines a favorable posture for the escape of fluids from the trachea and for the afflux of blood to the brain, with a ready method of artificial respiration. Duke places the infant face downward, its thorax resting upon the open palm of the left hand; the ribs are gently compressed by the other hand: the mouth is cleansed, and the finger passed down the pharynx to admit air. If this is not successful, the child is plunged into a hot bath. Richardson urges that the child's body remain quiet during efforts to establish respiration. The feeble condition of the heart strongly contraindicates violent disturbance to the child. The position of the body should be horizontal. Air introduced should be warmed to 90° F. Manual respiration by Silvester's or Hall's method is recommended, and Richardson describes an apparatus composed of a pair of bulbs by which air may be pumped into the respiratory passages. Two pieces of tubing are passed to the nostril, and a bulb upon one injects air, while a bulb upon the other favors the discharge of mucus and vitiated air.

He also describes a method of using a simple bellows in connection with a nasal tube. The treatment of asphyxia by tracheotomy is seldom successful; there is rarely an impediment in the respiratory passages of the child which cannot be overcome by the introduction of the catheter.

In reviewing the treatment of asphyxia we desire to call attention to the pathology of the affection and to the relative value of different methods of treatment. The removal of mucus from the nostril, trachea, and bronchial tubes can be most readily effected by suspending the child in an inverted position; this favors also afflux of blood to the medulla and respiratory centre. Gentle, passive respiratory movements should be employed, but so conducted as to give the child the least disturbance possible. The return of the circulation and the reflexes should be eagerly awaited, and so soon as these phenomena are present the prognosis becomes much more favorable. The warm bath and the application of a mild counter-irritant—cold water, spirits, simply a current of air from bellows directed against the epigastrium—usually cause respiratory movements. In strong children, when the reflexes are present and the heart-beat becomes perceptible, Schultze's method, practised gently for a short time, is of value. Should the circulation fail, it is admissible to inject hypodermatically $\frac{1}{100}$ of a grain of strychnia and a few minims of tincture of digitalis. If mucus is not expelled by the inverted position, the use of the catheter with suction and insufflation is advisable. When respiratory efforts have become established, but repeatedly fail, a mild faradic current of electricity and the inhalation of oxygen under pressure are of decided value. One pole of the faradic battery should be placed at the back of the neck, and the other over the thorax and alternately over the epigastrium. Bonnaire obtained good results in foetal asphyxia by inhalation of oxygen—a procedure which we have repeated with like good results in foetal asphyxia and that of older children complicating pneumonia. As Lusk remarks, in cases of deep asphyxia patience, watchfulness, and a hopeful spirit are prerequisites of success.

Following asphyxia, the infant is exposed to danger of death from inanition, and, as has been stated, from catarrhal pneumonia. The use of the incubator is of especial value in maintaining the circulation in these cases, and favoring the gradual expansion of the lungs if atelectasis be present. Winckel has obtained good results from the permanent hot bath at a temperature of 98.6° to 100° F. every twelve to twenty-four hours. Such children are fed every two hours. The bowels are promptly emptied by rectal injections. Winckel has devised a bathtub for such cases, an illustration of which is appended. We add also an illustration of a modification of Auvard's incubator, which we have used successfully in the Philadelphia Hospital and in the Maternity Department of the Jefferson Hospital.

The interior of the box is divided into two parts by an incomplete horizontal partition, placed about six inches above the bottom of the box. In the lower part, which is intended for hot cans, two openings are necessary—one at

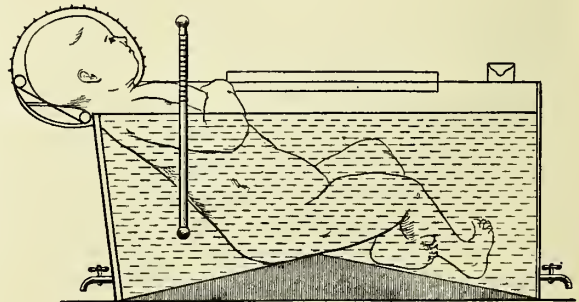
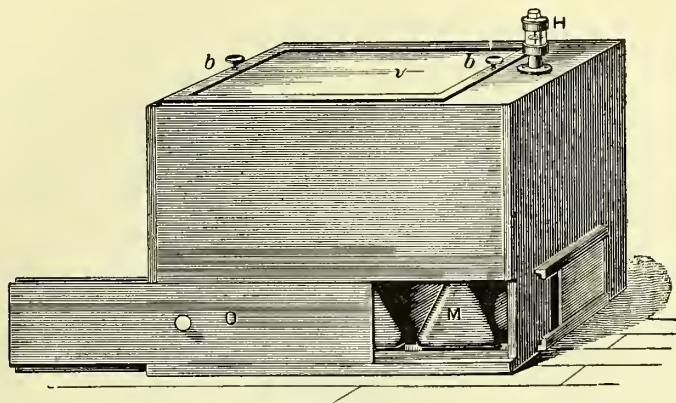


FIG. 3.

The Permanent Bath. (Winckel).

the side, occupying the whole length of the side, closed by a sliding door opened at pleasure from either end, as a means of placing the hot cans. The

FIG. 4.

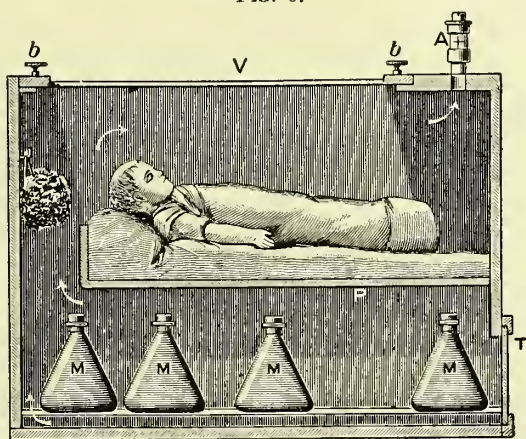


Incubator.

b, b, lid with glass plate; *v*, glass plate; *H*, ventilating tube; *O*, slide closing hot-air chamber; *M*, hot-water cans.

other opening is at one end of the box, closed by a door not fitting tightly, to admit a small amount of air. The upper part, arranged to receive the infant,

FIG. 5.

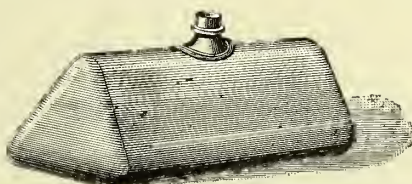


Interior of Incubator.

is covered on top by a plate of glass, fitting completely, with two buttons or knobs to admit of its being easily raised. On the top is also arranged a small metal tube containing a small rotary fan very easily moved by a weak current of air. In the opening where the two compartments join a sponge is placed, wet with water to humidify the air, and a thermometer by which to regulate the temperature.

Cases are not infrequently met with where death occurs soon after labor with

FIG. 6.



Hot-water Can for Incubator.

symptoms of partial asphyxia: a clear diagnosis is often impossible, until post-mortem examination reveals partial heart-clot, syphilis, atelectasis, or lobular inspiration pneumonia as the cause for this mortality.

HÆMORRHAGES FROM MUCOUS SURFACES.

The new-born infant often presents hæmorrhages from mucous surfaces of the body. Among the most frequent of these is a discharge of blood from the vagina, occurring at birth and persisting afterward. An examination of the mucous membrane in these cases frequently detects a condition of capillary granulation which bleeds easily upon the slightest movement of the child. In a case recently under our observation at the Maternity Department of the Jefferson College Hospital an ill-developed female child presented this phenomenon at birth. A blood-count made of this child, and compared with that of a healthy infant, shows the following:

Healthy Child.—Red corpuscles per cubic millimetre, 5,450,000, by counting forty squares (Thoma-Zeiss hæmocytometer). White corpuscles per cubic millimetre, 11,000. Proportion of white to red, 1:495. Hæmoglobin, 65 per cent. of normal. Blood-plates by objective, blood prepared by means of Hayem's solution: the number was much less than the usual amount, which should be about 250,000. The red corpuscles were irregularly formed, some crenated, some small and granular, others apparently rolled or turned upon themselves, resembling very much a bread roll. While this irregularity existed, their appearance was that of normal corpuscles, and the percentage of hæmoglobin (65) proved them to be almost normal. In children the percentage of hæmoglobin is not so great as in adults; in the young or in any case where the growth is rapid the red corpuscles are always irregular in appearance, which is not at all indicative of disease. The slight increase in red corpuscles is normal to the new-born. (Plate II. Fig. 1.)

Anæmic Child.—Red corpuscles per cubic millimetre, 2,000,000. White corpuscles per cubic millimetre, 12,000. Proportion of white to red, 1:166. Hæmoglobin, 35 per cent. of normal. By careful examination no blood-plates could be found. In this case the red corpuscles were irregular, crenated, granular, and many disintegrated. By actual count this specimen would give over five million red corpuscles per cubic millimetre, but counting normal corpuscles would give only two million. The object of the count being to know the number of oxygen-carriers per cubic millimetre, it would give a wrong idea to enumerate those disintegrated and diseased corpuscles. There was a slight increase in the number of white cells, but their appearance was normal.¹ (Plate II. Fig. 2.)

The condition underlying such hæmorrhage is that of anæmia or malnutrition of the blood, with resulting ecchymoses. In parts accessible to treatment, as the mouth, vagina, rectum, or bladder, injections of hot dilute creolin solution or boracic solution are indicated. Treatment of the anæmia, however, by administration of food, by arsenic, inunctions with oil, and the administration of olive or cod-liver oil will result in gradual recovery.

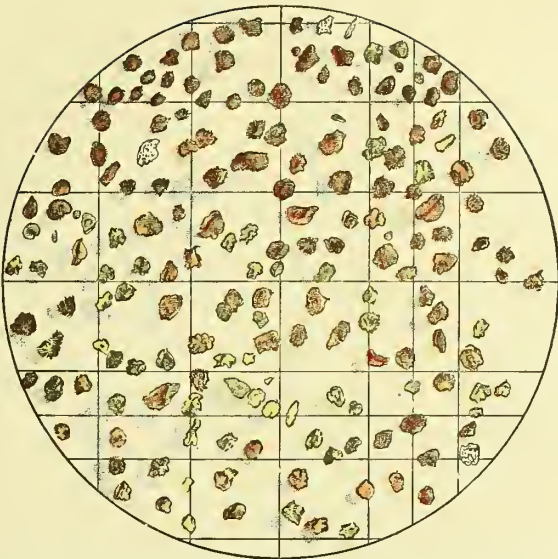
OBSTETRIC PARALYSIS AND INJURIES TO THE NERVOUS SYSTEM.

Direct injury to the nervous system received during birth has long been recognized as among the dangers to which the infant is exposed. Paralysis of

¹ For the examination and description of the blood in these cases I am indebted to Dr. D. B. Kyle, Instructor in the Examination of the Blood in the Jefferson Medical College. Dr. W. H. Wells, one of the physicians to the Jefferson Maternity, has prepared the drawings illustrating the appearance of the corpuscles.

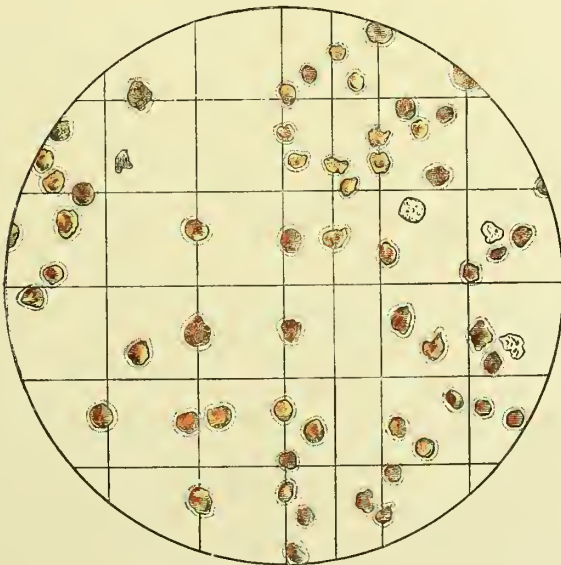
PLATE II.

FIG. 1.



Blood of Healthy Child one month old. Hæmoglobin normal. Drawn from Thoma-Zeiss Hæmocytometer. Objective $\frac{1}{8}$ Reicherts. Blood-count by Dr. Kyle; drawing by Dr. Wells.

FIG. 2.



Blood of Anæmic Child suffering from Hæmorrhage from Mucous Membranes. Total corpuscles, 5,000,000, of which 2,000,000 were normal. Blood-count and drawing as in preceding figure.



the facial nerve caused by pressure with the forceps upon the nerve at its foramen of exit often follows instrumental delivery. The brachial plexus is also frequently injured by the same agent. Hemiplegia, idiocy, and impaired cerebral development have been ascribed as consequences of injury received at birth. The view previously held, that the forceps is a valuable agent for compressing the foetal head and exercising leverage and forcible rotation, has given place to the belief that the forceps is essentially a tractor, and that the mechanism of rotation depends upon the relation in size and symmetry between the head and the pelvis, and, as well, the resistance of the pelvic floor. Murray has shown by experiment and clinical observation that the foetal skull is compressible in an antero-posterior direction by the sliding of the occipital and frontal bones under the ends of the parietal bones. This compression is not accompanied by any appreciable increase of the transverse diameter. The antero-posterior shortening is compensated for by a vertical elongation of the skull, providing for the accommodation of the cranial contents. These conclusions are, however, based upon the employment of axis-traction, without which such compensatory elongation cannot be confidently assumed. Murray was also careful to avoid forcible traction. Under such circumstances it may be held that moderate pressure with forceps, resulting in compensatory elongation of the vertical diameter of the foetal skull, need not be expected to cause paralysis, hæmorrhage, or fracture. This pressure, however, must be gradually applied, and traction made in the axis of the pelvis; otherwise a portion of the head will be forced against the promontory of the sacrum, and injury must result. When gentle axis-traction fails to cause the head to descend, a diagnosis of disproportion between the head and the pelvis should be made, and efforts at forcible delivery should cease.

The results of injudicious delivery with forceps are well illustrated by Lane. A boy sixteen years old, delivered at birth with forceps, exhibited a groove three and a quarter inches long from the right coronal suture to the lambdoid; the floor of this groove seemed one-fourth of an inch below the scalp; the left arm was weaker than the right, and its movements defective. The left leg was weak. Reflexes were exaggerated and clonus was present. The depressed portion of bone was raised; the bottom of the depression encroached upon the area of the skull. Prompt amelioration of the epilepsy followed. Duchenne, Gueniot, De Paul, Rogers, and others have described injuries to the brachial plexus caused by forceps and by manual extraction of the child. Erb has clearly described injuries to the brachial plexus accompanying delivery in breech presentation. Hoedamaker describes injury to the fifth and sixth cervical nerves resulting from delivery in breech presentation when the arms become extended above the head. Feriberg describes a case of paralysis caused by pressure upon the brachial plexus during delivery after version; paralysis was but temporary, the patient subsequently making a good recovery.

The medico-legal aspect of injuries to the new-born child requires the differentiation of lesions received during birth by forceps or the pressure of the mother's pelvis, and injuries occurring by precipitate labor without assistance or by the wilful act of the mother or an accomplice. Dittrich reports cases of depression in foetal bone, bounded by a well-defined ridge, following application of the forceps in cases of contracted pelvis. Küstner describes funnel-shaped depressions in the foetal skull following forcible delivery by forceps. Von Hofmann has found a spoon-shaped depression the most frequent form of lesion in a considerable number of cases. Fracture of the orbital region of the skull has been observed by Lihotzky to follow forcible forceps delivery. Rupture of a meningeal vein and death from hæmorrhage have been observed and

reported by Koffer in the clinic of Gustav Braun. Kundrat reports an interesting case of depression upon the parietal bone of a new-born infant, with cerebral hæmorrhage, in which the evidence seemed to show that the lesion was caused by direct violence on the part of the mother after the birth of the child. Von Hofmann has further drawn attention to injuries to the foetal cadaver which might occasion suspicion of intentional violence during birth. Naturally, defects in the ossification of the skull may result in lesions accompanying normal labor and simulating injuries at birth.

Fritsch describes the characteristics of injuries caused by precipitate labor, the child falling upon the floor or ground, to be as follows: The fracture begins in a suture, and extends outward to the middle of the bone; usually there is but one fissure, which ends where the bone is thickest. The parietal bone is most often affected, the fissure ending in the parietal eminence. As a general distinction, it is to be observed that direct violence is accompanied by hæmorrhage; that injuries examined immediately after birth, where fracture occurs, show frequently a well-defined border to the lesion, which tends to grow less sharp in contour if the child survives. Kundrat also lays stress upon the relative breadth of the sutures as a factor in influencing hæmorrhage during birth.

A most interesting question arises as to the bearing of these injuries upon the future health and development of the child. Osler found, in the records of the Philadelphia Infirmary for Nervous Diseases, 9 cases of paralysis following forceps delivery; in 6 of these it was reported that the forceps injured the child: some of them had scars following labor. In all cases the paralysis gradually appeared within a short time after labor. M. Allen Starr describes cases of brain-atrophy manifesting itself in hemiplegia, mental defects, and sensory defects, accompanied frequently by epileptiform seizures, and resulting from congenital conditions or lesions occurring at birth. Sachs and Peterson in 49 cases of congenital cerebral palsy found 16 in which some difficulty in labor occurred. These statistics are now more comprehensive than those of Little and Gaudard, Wallenberg and Osler. Sachs and Peterson, however, include all forms of cerebral paralysis and of tedious labor as well as instrumental delivery. Sachs has expressed the opinion that prolonged labor does more injury to the child's brain than the proper application of forceps.

We have considered the prophylactic treatment of these conditions under that of the treatment of visceral hæmorrhage. The question arises, however, What shall be done in a case in which a child is born and survives with such an injury? Although we find no record that such a procedure has been attempted, yet the suggestion of Nancrede and other surgeons that depressed bone be elevated by surgical interference is certainly rational. We believe that where pressure-symptoms are present, or where the lesion is extensive and follows severe pressure, such should be the line of treatment. The success attained in operating immediately after birth upon cases of umbilical hernia gives encouragement to the belief that surgical interference in these cases is justifiable. It is interesting to note a superstition common among the laity in some quarters to the effect that the doctor by manual pressure and counter-pressure is expected to shape the head of the child during the first few days after its birth.

FRACTURES AND DISLOCATIONS OF THE TRUNK AND EXTREMITIES.

The skeleton of the foetus may be fractured while in the uterus. Such fractures, however, must be carefully distinguished from congenital malformation, which closely simulate fracture. Amniotic adhesions during the first and second months of intra-uterine life are the most frequent causes of these mal-

formations. An apparent scar is often present in these cases, and must be referred to precipitate flexion of undifferentiated layers in the embryo. Spurious callus may be present, caused by defective development of the bone, although the amount of callus is less than after actual fracture. Sperling would distinguish between malformation and fracture by the fact that in malformation the fingers and toes of the limb affected show defective development, while in fracture such defective development of fingers and toes is absent. Hodgen describes a foetal skeleton containing sixty-five fractures which he thinks were caused by muscular action during uterine life. He describes also, in a healthy child, a fracture of the clavicle, which was not discovered for several days after birth; the child was large and was delivered by forceps.

The most frequent fractures in the long bones are those of the clavicle, humerus, and femur. Fracture of the clavicle near its acromial end is occasionally complicated by severe injury to the brachial plexus, as illustrated in a case reported by Knight; permanent injury of the shoulder with paresis followed. Fracture of the clavicle is most frequently caused by forcible extraction of the shoulders.

Fracture of the humerus most frequently occurs in the delivery of the after-coming head when the arms become extended above the head. Fracture of the femur usually results from difficult version and extraction. Fractures of the bones of the leg, of the ribs and sternum are rarely met with, and only in cases of forcible extraction through highly-contracted pelvis.

Dislocations of the foetal skeleton are frequently confused with fracture, and are caused by the same manipulations which give rise to solutions of continuity. Dislocation and separation of the epiphyses of the humerus at the elbow-joint have been not infrequently observed after manipulation.

The treatment of fractures and dislocations of the trunk and extremities is based upon the principles of surgery commonly followed. Difficulty has been experienced in maintaining the fragments in apposition by reason of restlessness in the child, and the necessity to move it frequently when it nurses and when it is cleansed. Fractured clavicle will heal without deformity with a very simple retention dressing if the infant be kept assiduously upon its back. Fracture of the humerus and of the femur may be treated to advantage by some form of splint material which can be dipped in hot water, moulded to the child's limb, and retained in position by a simple roller bandage. Firm and unyielding dressings must be avoided in these cases, as the danger of injury to the tissues by pressure is very great. Fractured ribs and sternum may be successfully treated by a broad flannel bandage pinned smoothly about the chest. Dislocations require the same principles of treatment which should be followed in managing fractures.

The prognosis in fractures of the foetus is usually good. As most of them are of the "green-stick" variety, a favorable result without deformity is the rule rather than the exception. When congenital malformation is present, the practitioner should be guarded in his prognosis. He may remedy webbed fingers and toes by dissecting them apart, but he will scarcely hope to see a congenitally malformed limb become perfectly developed.

UMBILICAL HÆMORRHAGE.

If the umbilical cord be tied firmly with an aseptic ligature after its pulsations have ceased, if the stump be powdered with boracic acid or salicylic acid 1 part to powdered starch 3 or 5, and if reasonable care be exercised to protect it from violence, hæmorrhage from the umbilicus or umbilical inflammation

rarely occurs. The cord may be best protected by enclosing it in a small mass of antiseptic cotton, directing the extremity of the stump upward and to the child's right, and pinning a flannel binder comfortably tight about the abdomen. In cases, however, where syphilis, hæmophilia, septic infection, and acute fatty degeneration, with hæmoglobinuria, are present, hæmorrhage may occur when the cord separates, or even before that time. This complication is not very frequent, Winckel having observed it but once in 5000 infants. Bouchut quotes Grandidier's analysis of 202 cases, from which he concludes that the hæmorrhage begins most often at night, and often accompanies colic, vomiting, somnolence, and jaundice, with ecchymoses of the skin. Bleeding occurs rather more frequently before the cord is entirely separated, and usually between the fifth and ninth days. The hæmorrhage takes the form of arterial oozing, the blood often failing to coagulate. The hæmorrhage may persist from one hour to several weeks. The mortality from umbilical hæmorrhage is estimated at 80 per cent.

The treatment is frequently futile. A needle, armed with a silk ligature, may be passed beneath the vessels and securely tied; two surgical pins may be passed beneath the bleeding tissue at right angles to each other, and the ligature may be looped around the pins. Pressure is indicated in treating umbilical hæmorrhage; it is best made with antiseptic cotton on which iodoform has been freely sprinkled.

UMBILICAL POLYP.

The umbilicus may fail to heal perfectly, and abundant granulations, bleeding upon touch, and polypoid growths may develop; they are best treated by the application of nitrate of silver or other suitable escharotic.

UMBILICAL HERNIA.

A protrusion of the abdominal contents may accompany defective closure of the umbilicus. While it is indicated to palliate this condition by suitable dressings, yet it has been found possible to secure a radical cure by operation very soon after birth. Runge describes a case operated upon successfully sixteen hours after birth. In the majority of cases a cure may be effected, in a period varying from one to six months, by the application of an umbilical button. This consists of a hard-rubber disk convex on the applied surface, which is held in position by a broad band of surgeon's adhesive plaster.

GASTRO-INTESTINAL HÆMORRHAGE.

This complication depends upon a purpuric condition, and manifests itself most frequently from the fifteenth to twentieth day after birth. Kiwisch reports cases of hæmorrhage from the intestinal tract following the normal birth of apparently well-nourished children. The first symptoms were discharge of blood and restlessness, occurring from twelve to thirty hours after labor. The abdomen became dull and tumid, the patients were pallid, and in some instances vomited blood; death ensued within forty-eight hours.

According to Grynfeldt, gastro-intestinal hæmorrhages usually take place during the first three days after birth (Rilliet, Silbermann, Dusser), though in a case of this author's it occurred on the fourth and fifth days, and in two instances, seen by Rilliet, the children were fifteen and twenty weeks old. Sex seems to play no special predisposing rôle, but the influence of morbid

antecedents in the parents appears to be a factor of some importance. Pinard, Champetier, Auvard, and others have noted syphilis in the progenitors, but this is regarded by Grynfeldt as only a cause acting indirectly in deteriorating the health of the parents. Hæmophilia has certainly been proven in some instances.

The pathogeny is quite as obscure as the etiology. The lesions observed at autopsies are the most variable. Ulcerations of the stomach and intestines have been found; again, only a simple congestion; while other cases have shown a complete absence of visible lesion. - Grynfeldt advances a theory suggested by observations of Billard, and confirmed by personal studies of the histology of the digestive mucous membranes of new-born infants. These show that the vascular supply of the mucous membrane of the stomach and intestines is exceedingly rich at this period of life. Adding to this state of physiological congestion a congestion or impeded circulation in the liver, he finds it easy to ascribe the cause of such hæmorrhages to exaggerated tension in the portal area. This view, he believes, is supported by the fact that these hæmorrhages, at first sudden and profuse, quickly cease, thus resembling a true depleting loss of blood.

The first symptom is usually the hæmorrhage itself. Blood flows from the mouth following efforts at vomiting, or from the rectum, more or less mixed with feces or in clots; quite often both phenomena are coincident, hæmatemesis being usually the earlier. When one alone occurs, hæmatemesis is by far the more frequent. In spite of the gloomy prognosis evidenced by the statistics of Dusser (43 deaths in 78 collected cases), a more hopeful view must be taken.

In treatment, tannin in syrup of rhatany offers an efficient astringent potion. One and a half to two and a half grains of ergotin in mucilage are employed with satisfaction by Widerhofer of Vienna.

ICTERUS NEONATORUM.

The physiological icterus of the new-born infant appears on the third or fourth day of life, is characterized by a yellowish pigmentation of the face and breast, persists for about a week, and does not seem to disturb the patient's general condition at all. The urine is dark in color, containing bile-stuff, while the stools lack the color usually given by their mixture with bile. The cause of such icterus is thought by Birch-Hirschfeld to be swelling of Glisson's capsule, commencing at the umbilical vein, and by œdema preventing the free discharge of bile through the hepatic vessels; hence the jaundice is hepatogenic. Hofmeier thinks icterus is caused by the enormous number of red blood-corpuscles which are formed in the liver and hinder the production and discharge of bile. The entrance of this coloring matter into the blood is furthered by catarrh of the duodenum and congenital stricture of the ductus choledochus. Halberstam found undissolved bile-stuff in the urine of children with icterus, and the epithelium of the kidneys infiltrated with the same coloring matter.

The harmless character of this jaundice and its spontaneous disappearance should not make it a subject of anxiety to the physician or parents; it sometimes is due to slight changes in diet or any temporary disturbance of the child's general surroundings. Beyond the regulation of the bowels by the most simple laxatives, no treatment should be employed for this condition. Infective jaundice will be considered under the head of infections which attack the fœtus.

THE INFECTIONS ATTACKING THE NEW-BORN.

The recognition of bacteria, ptomaines, and toxins as causes of disease has served to explain many disorders of the foetus and infant at birth not previously understood. Most frequent of these infections are those by the micrococci of gonorrhœa and the streptococci of suppuration. Gonorrhœa in the mother affords the best of grounds for fearing gonorrhœal infection in the new-born child. The most usual site of this infection is the conjunctiva, and ophthalmia neonatorum is a familiar sequence of maternal gonorrhœa. The treatment of this disorder will be considered in another section of this book. We are interested, however, in the practical prophylaxis of such infection: if the practitioner could be absolutely positive that the mother had never been infected by the gonococcus, prophylaxis would be entirely unnecessary. In hospital patients, however, there is always room for suspicion; and in private cases, although there may seem no adequate reason to fear such a complication, yet its appearance will often surprise and disappoint the attending physician. No information will be gained in this matter from interrogating the patient: if she has ever been infected, her husband has certainly not told her the cause of the disorder, and her physician may have kept her in like ignorance. Furthermore, in women who have never been infected by the gonococcus there occurs at the latter portion of pregnancy a vaginal discharge which is capable of setting up a mild conjunctivitis in the infant. Hence a practical rule may be followed to advantage, that where a vaginal discharge persists during the latter portion of pregnancy the use of antiseptic douches is certainly indicated. These douches may be, preferably, creolin or bichloride of mercury: the first has the advantage of impairing the natural condition of the mucous membrane of the vagina less than does the mercurial; it is also a safer substance to put in the hands of a patient. On the contrary, its odor is disagreeable to some, and when used in a strong mixture it causes considerable irritation and burning. In a strength of one teaspoonful to the quart the resulting mixture is seldom so irritating as to cause discomfort. The quantity used should be not less than a quart, and the douche should be preferably taken while the patient is in the recumbent posture. The douche-bag should hang not higher than three feet above the patient's body, and the force of gravity alone should be employed in giving the douche. If bichloride of mercury be chosen, 1 : 5000 is sufficiently strong for such use.

In patients admitted to hospitals, suffering from the effects of previous gonorrhœa or having acute gonorrhœa, the treatment must be more radical; here a preliminary thorough cleansing of the vagina should be made with green soap and creolin, the mixture containing 2 per cent. of the creolin: following this, creolin douches, four times in twenty-four hours for the ten days preceding labor, will be found of advantage. Should the mucous membrane not tolerate such frequent douches, the vagina may be tamponed with iodoform gauze containing 50 per cent. of iodoform, and the number of douches be reduced one-half. In all hospital cases a preliminary douche of green soap and creolin may be used to advantage; in private practice a preliminary douche of bichloride, 1 : 5000, may also be employed to the advantage of mother and child.

Aside from ophthalmia, gonorrhœa may infect the infant at birth upon other mucous membranes. Rosinski describes the results of interesting investigations made by him upon gonorrhœa occurring in the mouths of new-born infants. The lesions caused by this germ in the mouth develop only where the pavement epithelium has been removed. These cells are especially fragile

PLATE III.

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FIG. 1.

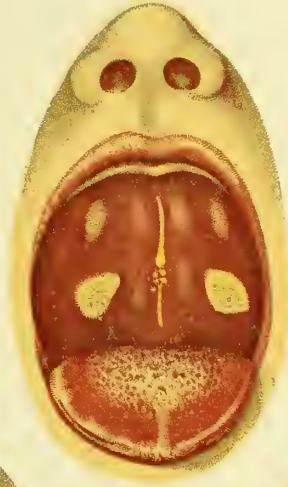


FIG. 2.



FIG. 3.



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FIG. 4.



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Gonorrhœa of the Mouth in the Newborn (Rosinski).

in the young child, and hence the readiness with which infection occurs. It is interesting to note that in gonorrhœal ophthalmia it is very rare to find that the lachrymal sacs become involved; it is also true that the cylindrical epithelium of the naso-pharynx seems also to resist successfully invasion by the gonococcus. Clinical observation shows that these cases develop usually between the fifth and tenth day of life, resulting often from infection from the genital canal occurring at birth, and oftentimes through direct infection at the hands of attendants. This is especially true where the epithelium of the mouth is destroyed through efforts at cleansing. These cases are remarkable for the fact that they affect the general health so little; the children nursing well and seeming free from pain. The lesions are yellowish plaques, surrounded by a border of pale-reddish tissue, in which the process of healing usually begins upon the third day by a reaction zone of deeper color. The epithelium is renewed from the borders of the plaque, pus-cells being thrown off as the healing progresses. Scar-tissue is never developed in these cases. The accompanying plate gives an excellent idea of the appearance of the lesions. (Plate III.)

The treatment of gonorrhœa affecting the mouth of the new-born consists in careful avoidance of injury to the epithelium; the finger should not be inserted into the mouth of an infant suffering from this disorder: the affected surfaces should preferably be sprayed with a solution of hydrogen peroxide or a saturated solution of boracic acid. Such treatment is usually amply sufficient to secure the recovery of the patient. The infant's general condition often requires attention in these cases, and its food and hygiene are matters of great importance.

GENERAL SEPTIC INFECTION.

Streptococci, bacteria, and ptomaines of septic infection usually find entrance to the foetal body through the granulating surfaces upon the umbilicus; the result is arteritis and phlebitis of the umbilical vessels, resulting in the formation of thrombi and the infiltration of the surrounding tissues with bacteria and ptomaines. Both umbilical arteries are usually involved, the infection extending from the umbilicus to the bladder. The umbilical ring may ulcerate, or may have healed entirely while the infection has proceeded within the abdomen. According to Weber and Runge, the tissue about the arteries is usually first involved; the iliac vessels and the retroperitoneal connective tissue usually escape; in two-fifths of cases Runge found pneumonia or pleurisy with small metastatic abscesses. Peritonitis and pyæmic metastases in the abdominal viscera and the joints have also been observed. In umbilical phlebitis the capsule of the liver and the liver itself become involved. Pericarditis, pleuritis, and other pyæmic complications are often present. The symptoms of such infection are often obscure. The umbilicus may become inflamed shortly after birth; the child has fever, is restless, holds its legs and thighs flexed, and often becomes jaundiced. Death may occur in convulsions, but occasionally recovery ensues. The treatment of umbilical septic infection is largely prophylactic: thorough antisepsis as regards the physician, nurse, and external genital organs of the patient, a suitable and cleanly dressing for the umbilicus, such as previously given, and scrupulous cleanliness while the cord is drying and becoming separated, render umbilical septic infection a rarity. If the child be too feeble to have the full bath for the first month of life, it is comparatively easy to allow the cord to remain undisturbed. Where, however, the child is bathed daily in the bath-tub, such of the cotton as may

become wet should be carefully removed, the cord repowdered, and fresh cotton applied.

The constitutional treatment of an infant suffering from septic infection through the umbilicus consists in the reduction of excessive fever by judicious sponging with warm or cool water, and the free administration of dilute alcohol and nourishing food. While quinine, if it can be taken, is a useful auxiliary, yet alcohol is the drug of most importance for such cases. Infants suffering from severe infections often bear strychnia as a stimulant better than might be expected from theoretical considerations only.

ERYSIPELAS.

The micrococcus of Fehleisen may obtain an entrance at the umbilicus, and erysipelatous inflammation of the subcutaneous and cutaneous tissues may result. This process may go on even to the extent of gangrene and sloughing of the affected parts. Cases of mixed infection resembling erysipelas may develop, complicated by diphtheria, as in illustrative cases reported by J. Lewis Smith from the records of the New York Infant Asylum. The infection may localize itself in multiple abscesses beneath the skin, or, extending to the peritoneum, may cause death from acute peritonitis.

The treatment of erysipelatous infection of the umbilicus and surrounding parts consists in thorough applications locally of peroxide of hydrogen, boracic acid, or thymol solution, 1 : 1000. Following this, equal parts of iodoform and boracic acid may be employed freely. When pockets of pus form, they should be promptly opened with a knife or scissors and thoroughly douched with an antiseptic. The child's general strength must be assiduously supported by alcohol, food, and strychnia or quinine. As a stimulant in severe prostration, hypodermatic injections of camphor in oil, or administration, by the mouth, of freshly-made English breakfast tea, with rum, will be found of service in some cases.

ACUTE PERITONITIS IN THE NEW-BORN.

Acute peritonitis occasionally arises very soon after birth as a complication of erysipelas or from some pathological process developing in the intestine. The communication in lymphatic channels between the intestine and the peritoneum seems unusually free in the infant, and as a result peritonitis rapidly supervenes. Cassell describes three interesting cases of this sort. Lorain, Quinquaud, and Silbermann have also reported illustrative cases of this disorder.

TUBERCULAR AND TYPHOID INFECTION.

There exists certain ground for belief that the foetus *in utero* may become infected by tubercle bacilli and also by the bacilli of typhoid. The first few days after birth may witness acute miliary tuberculosis or the development of a well-marked typhoid condition. As regards the former, the usual clinical signs of acute tuberculosis will be present: it must be remembered, however, that the infant rarely survives acute tuberculosis long enough for the formation of lung-cavities, and hence physical signs will often be lacking. The character of the fever, the rapid, uninterrupted course of the disorder, with increased dullness over the thorax, and the development of harsh and bronchial breathing, will usually enable the physician to make a diagnosis.

While treatment up to the present time has been practically unavailing, it is of interest to note the experiments of Pinard in using injections of the serum of dog's blood in these cases ; in a series of twenty-one infants so treated he believes that benefit has resulted, the remedy seeming to act as a powerful tonic and stimulant.

The intra-uterine transmission of typhoid infection is well illustrated by a case recorded by Giglio. The presence of the typhoid germ was demonstrated in the tissues of an apparently normal fœtus and placenta born forty-six days after the beginning of typhoid fever in the mother.

The treatment of typhoid in the new-born is practically that in the adult, reference being had to the ease with which the infant is stimulated or depressed. The prognosis in such cases is exceedingly grave.

INSPIRATION PNEUMONIA.

In prolonged labor, complicated by a septic condition of the mother's birth-canal, premature inspiratory movements on the part of the fœtus may result in the inspiration of septic material: lobular septic pneumonia may result, and, occurring soon after birth, frequently proves rapidly fatal. Here, again, the efforts of the physician lie in prophylaxis, in delivering the patient promptly, and maintaining so far as possible an aseptic condition of the birth-canal until labor shall terminate.

TETANUS.

The infant may become infected with tetanus, and this disorder may appear in well-marked type from the sixth to the ninth day after birth. The tetanus bacillus usually finds its entrance at the unhealed umbilicus. Brieger has shown the specific cause of this disorder, and Beumer and Peiper have confirmed by clinical observation the identity of trismus and tetanus of the new-born with inoculative and wound tetanus. The mortality among infants is exceedingly large, and recovery is the rare exception. Appearing with symptoms of restlessness, night-terrors, and frequent cries, the child often becomes nauseated, has slight diarrhœa, and is then attacked by trismus. This, at first intermittent, finally becomes persistent, and develops into tetanic contractions of the entire body. Icterus is usually present. The disorder rarely lasts more than three or four days, the child perishing in collapse from twelve to twenty-four hours after the beginning of the convulsions. High temperature is usually present at the time of death. On post-mortem examination effusion of blood and serum in the cerebral tissues is frequently found. The violence of the convulsions may give rise to hæmorrhages into the muscular interspaces or into the tissues of the mediastinum.

In treatment hydrate of chloral and alcoholic stimulants give most prospects of relief. Holt has reported a case which recovered under the free use of bromide of potassium. A specific method of treatment by the injection of a substance similar to tuberculin has not, so far as we know, yet been employed in this disease. There would certainly seem to be reasons for testing its value.

MASTITIS.

Mastitis in the new-born infant is to be regarded as a mild septic infection when the disorder comes to the point of suppuration and phlegmonous inflammation. The mammary glands of new-born children frequently become engorged and tender, but this condition subsides if the glands be let alone and

protected from external violence. When, however, infection occurs, pus-formation may take place and a septic mastitis may result. Such a complication, however, is exceedingly rare where antiseptic precautions are habitually taken in the treatment of labor cases. A distinction must be made clinically between simple engorgement of the breast and infection. In the former the child's temperature remains but little disturbed, its appetite is unimpaired, its rest remains practically as before. If the glands be carefully but gently washed with soap and water and bathed with bichloride, 1 : 10,000, a thin layer of absorbent cotton put over them, and a soft flannel bandage pinned snugly about the breast and supported over the shoulders by shoulder-straps or some other simple device, the glands may remain undisturbed for several days unless fever or restlessness indicates inflammation. On the other hand, where infection is present and pus has formed, prompt emptying of the gland by incision, with disinfection of the cavity, is indicated.

INFECTIONS OF THE BLOOD.

Profound alterations of the blood and nutritive cellular processes in the new-born, the probable result of infection at birth, have been described under various names by different observers.

Hecker and Von Buhl describe a disorder of infants born in asphyxia characterized by cyanosis, vomiting, icterus, profuse parenchymatous hæmorrhage, accompanied by acute fatty degeneration of visceral epithelium and heart-muscle. Phosphorus- and arsenic-poisoning were excluded in diagnosis, and the malady was named "acute fatty degeneration of the new-born," or Buhl's disease. Its pathology is not perfectly explained, but it may be classed among the infective disorders resulting in the extensive disintegration of the blood.

Acute hæmoglobinuria of the new-born was first clearly described by Winckel, who reported twenty-three cases of the disorder. It is characterized by swelling of Peyer's patches and the mesenteric glands, blackish-red staining of the pyramids of the kidneys, with stripes of hæmoglobin coloring, fatty degeneration of the liver and other viscera. Hæmatogenic icterus is present, the hæmoglobin being extensively changed into bilirubin. The urine is dark brown-reddish in color, contains hæmoglobin, epithelium, casts, and micrococci. Chemical poisons as a cause were excluded in diagnosis. The mothers showed no infection, the children were usually well developed. The mortality was 19 out of 23. The cause of the disorder is not clearly demonstrated. It is undoubtedly an infection which attacks the blood, resulting in hæmoglobinæmia. Prophylaxis and treatment, beyond the faithful employment of antiseptic precautions, are practically without avail.

Hæmatogenic jaundice, accompanied with multiple oozing of blood, has been recently described in an interesting paper by Partridge. In the case reported recovery ensued. In 1166 infants born at the Nursery and Child's Hospital, New York, 11 cases of hæmorrhage occurred, with a mortality of 75 per cent. At the Sloan Maternity Hospital, in 850 patients there were 14 cases; mortality over 60 per cent. No intelligent family history of bleeding was obtained.

Somewhat similar to these cases are those of the disorder known as

MELÆNA NEONATORUM.

Infants dying with profuse hæmorrhage from the stomach and intestine have revealed an ulcer of the duodenum as a cause. In explaining these phenomena

Landau assigns as a cause thrombosis of the umbilical vein, resulting in embolism in the vessels of the stomach and duodenum. Persistence of the ductus arteriosus and hæmophilia also have been assigned as causes. Kundrat in examining Winkel's case found excessive secretion of the gastric juice, which had partly digested the mucosa of the intestine and occasioned hæmorrhage. In other cases bloody stools and vomiting of blood persisted for several days. Recovery occasionally ensues.

The prognosis is exceedingly grave, and treatment is practically unavailing. The milder preparations of iron may be given by the mouth, and hot or cold applied to the surface of the body as the condition of the child indicates. An abdominal compress may also be useful.

In closing this consideration of the infective disorders of the new-born we must again emphasize the fact that while we are not, in the present stage of our knowledge, in a position to particularize regarding the precise nature of the infective agent and its mode of operation, still, the fact remains reasonably proven that these cases result from some direct infection occurring just before or during birth. It remains, then, the positive duty of the practitioner to see to it that rigid asepsis—and, better, antisepsis—is employed regarding his hands and instruments, those of the attendant, and also the external organs of the patient. Ehrendorfer, writing upon this subject, draws attention to the dangers of infection, not only from mother to child, but from one child to another in hospital wards. The practice of putting a number of children in the same crib is objectionable, as is the custom of bathing a number of children in the same bath-tub, and, still worse, of using the same towels or cloths for a number of baths. From the moment of birth each infant should have its own toilet appliances, be they of the simplest description. In cleansing the child absorbent material which can be thrown away and not used a second time is preferable. Separate vessels for bathing the child's body and for washing the head and face are also desirable. In this way septic matter from the umbilicus is kept away from the mouth and eyes, and *vice versâ*. Nurses may be drilled to advantage in these niceties in the care of infants, which are not simple matters of æsthetic neatness, but are founded upon pathological facts.

PART II.

THE DIATHETIC DISEASES.

LITHÆMIA.

BY B. K. RACHFORD, M. D.,

CINCINNATI.

LITHÆMIA (*λίθος*, stone; *αἷμα*, blood) is a term which was introduced by Murchison to designate a group of symptoms which he thought to be due to an excess of uric (lithic) acid in the blood. Austin Flint, Sr., used the term uricæmia for the same purpose. Alexander Haig and others have written largely upon the subject under the name uricacidæmia. A number of recent writers have grouped the same set of symptoms under the title lithuria. Concealed gout and American gout have also been very largely used in naming the same clinical manifestations. The writer has made a number of contributions to this subject under the title leucomaine-poisoning. All of these terms have found their way into medical literature, and all of them are more or less inaccurate. The term lithæmia heads this chapter not because of its propriety, but rather because of its long and widespread use by medical writers in describing a condition which is known by its symptomatology rather than by its pathology. We know that lithic acid is not responsible for all, or even the greater portion, of the symptoms of lithæmia. This term is therefore a misnomer and conveys a false idea of its pathology. Yet it is my belief that the time for rechristening this disease must await a fuller knowledge of its pathology than we have at present. Lithæmia is essentially an auto-intoxication resulting, as I believe, from the presence of an excess of the alloxuric bodies in the body media. Uric or lithic acid, from which the disease is named, is one of these bodies, and xanthin, hypoxanthin, heteroxanthin, and paraxanthin are the other important members of this group. The relative importance of these bodies as disease-producers is not at the present time clearly made out, and need not therefore further engage our attention.

Etiology.—*Heredity* holds first place among the etiological factors of lithæmia. In fact, one may say that this disease as it occurs in infants and children is essentially an inheritance from lithæmic ancestors.

An excess of proteid food may be a factor in developing lithæmia. It is believed that the alloxuric bodies have their origin either directly or indirectly from the proteid food. The more proteid food, therefore, the body is called upon to metabolize, the more of these waste products will be formed.

Inactivity will predispose to lithæmia. This factor is especially potent

when associated with an excessive intake of proteid food. It is probable that sedentary habits increase the liability to lithæmic attacks by furnishing diminished opportunities for the oxidation of the poisonous alloxuric bodies, since it is a recognized fact that these bodies, however they may be formed, may, under favorable conditions, be oxidized into non-toxic uric acid and urea. Active exercise in the open air, by furnishing the most favorable conditions for the oxidation of these bodies, will diminish the dangers of auto-intoxication.

Excretion of the Alloxuric Bodies.—The alloxuric bodies are excreted by the kidneys, the skin, and the intestinal canal. In this work the kidneys play the most important rôle. These bodies are removed by the kidney cells from the blood into the urine. Their presence, therefore, in great excess in the urine means that immediately before they were in solution in excess in the blood. Disease of the kidneys may cause an abnormal retention of these bodies in the blood. The excretion of these bodies by the skin is of especial importance when the kidneys fail to do their part of the work. The undoubted value of many of the hot springs in the treatment of lithæmic conditions depends upon the fact that the hot alkaline bath promotes the cutaneous elimination of the alloxuric bodies. In the hot months the skin is more active than in the cold months, and this may be one of the explanations of the comparative infrequency of lithæmic attacks in summer. The intestinal canal is a most important channel by which an excess of the alloxuric bodies may be eliminated from the blood and the tissues. In practice one often finds it necessary to call upon the intestinal canal to assist the skin and kidneys in the excretion of these bodies.

Symptoms.—In order to avoid confusion by the mingling of symptoms from totally different causes, I shall speak first of the symptoms which are thought to be due to uric-acid deposits in the urinary passages. The newly-born lithæmic infant is prone to eliminate an excess of urates in the first days of life. In such infants uric-acid crystals may be precipitated into the tubules of the pyramids of the kidneys and cause thereby much pain and irritation. These uric-acid infarctions may subsequently be washed out of the tubules and serve as the nuclei of urinary calculi. Jacobi says the vast majority of renal and vesical calculi have their origin in this way.

Quite recently I saw an infant two days old. It was crying bitterly, and seemed to be in great pain: its temperature was 104° F., and had been nearly that high for twenty-four hours. I learned that this infant had been born of lithæmic parents, and that it had passed urine but once since birth. The urine passed at that time was small in quantity and tinged with blood. As treatment it was given a warm bath, a cathartic, and water to drink. Two days later it was convalescent, with the renal secretion established. The urine passed by this infant on the third day deposited a red sand of urates on the diaper. This case is typical of a class of cases which represent the earliest manifestations of infantile lithæmia. When fever and long-continued paroxysms of crying occur in newly-born infants coincident with the passage of urine so heavy with urates as to deposit a red sand on the diaper, one is justified in making the diagnosis of this special uric-acid type of lithæmia. These lithæmic infants may, as they grow older, continue to suffer from attacks of painful urination accompanied by an elevation of temperature and irritation of the external genitalia. The paroxysms of crying which occur during and immediately following the passage of urine are very characteristic. In the interval between these fits of crying the child is fretful, and grows more so as the time approaches when it can no longer resist the inclination

to urinate. The urine is acid and contains an excess of urates and oxalates. In some patients it is so irritating as to cause a vulvo-vaginitis in the female infant and urethral irritation in the male. The clinical picture here presented is by no means peculiar to infants and children. In adults it is also common to find frequent and painful urination associated with the passage of urine small in quantity, high in specific gravity, and heavy with urates.

Nocturnal incontinence of urine in children may be a lithæmic symptom resulting from the irritable condition of the urinary passages and the instability of the spinal nerve-centres that not uncommonly occurs in these children. If one recognizes the fact that lithæmia is at times an important factor in producing incontinence of urine, one will succeed in curing cases of incontinence that have resisted other forms of treatment. I wish to note, however, that lithæmia does not rank among the most common causes of this neurosis.

True arthritic gout, resulting from uratic deposits in the tissues about the joints, is very rare in childhood, and moreover does not come within the scope of this paper.

The symptoms and treatment of urinary gravel are elsewhere described in this book.

With this outline of the rôle that uric acid plays in the symptomatology of lithæmia we may pass to the consideration of those symptoms of lithæmia which in the present state of our knowledge cannot be attributed to uric acid. The writer believes that these symptoms are the result of auto-intoxication caused in part, at least, by the alloxuric bodies other than uric acid.

Gastro-enteric Symptoms.—The gastro-enteric symptoms of lithæmia in infancy and childhood are little understood, and they are of vast importance. The history of the following cases, which are extreme examples of this type of lithæmia, will best serve to emphasize these symptoms:

Case A.—Male infant, eight months of age; has a gouty ancestry on both sides. This infant has had since he was two months old, at intervals of four to six weeks, the most characteristic lithæmic attacks. These attacks commence with nausea and vomiting, and very soon the infant refuses, and the stomach rejects, all food. The nausea and vomiting continue for from two to four days, and during this time nothing is retained by the stomach. These symptoms are accompanied by fever and by very rapid breathing, which is not explained by any pulmonary condition. The odor of the breath is sickening, the bowels are constipated, and toward the close of the attack the baby is prostrated and emaciated to an alarming degree. Accompanying and immediately following these attacks the stools are very putrid and sometimes oily in character. These lithæmic paroxysms come and go without apparent cause. They are quite independent of the wholesomeness and digestibility of the food, and the duration of the attack is but slightly influenced by medication.

Case B.—Age four years, a brother of infant A; has been having very similar lithæmic attacks since he was an infant a few months old. His attacks were formerly characterized by obstinate constipation, with fever, nausea, vomiting, and rapid breathing. The nausea and vomiting would continue for three or four days, and would then disappear as suddenly as they came, leaving the patient to slowly convalesce during the next few days. These attacks came and went without apparent cause. The mother soon learned to expect them every six or eight weeks, and also learned that they were self-limited. The point of special interest in this boy's case is that recently these attacks have changed in character. At the present time vomiting is no longer a prominent symptom. They are now characterized by headache with nausea, and followed by a more or less prolonged narcotism, during which the child falls into a deep sleep from which he awakens somewhat improved. In brief, one may say that the gastro-intestinal paroxysms of his infancy are being transformed into true migraine. This substitution of one form of lithæmic paroxysm for another is quite characteristic of the disease.

The disease may manifest itself in young infants by attacks of gastric pain, associated with rapid breathing, nausea, vomiting, and fever. The

gastric paroxysms may be so severe that all food is rejected for a period of from one to five days. The temperature may reach 104° or 105° F., but sometimes in the most severe cases the fever ranges between normal and 102° F. In these attacks the patient may be prostrated to the last degree, occasionally having a subnormal temperature. Toward the close of these acute attacks the infant or child may be much emaciated.

Occasionally these lithæmic paroxysms are ushered in by convulsions, which may recur with such regularity as to become quite characteristic symptoms of such attacks. These gastric paroxysms are self-limited. In duration and severity they are influenced but slightly by medical treatment. The nausea and vomiting go almost as quickly as they came, but there is left more or less abdominal tenderness and gastro-intestinal irritation, from which the infant or child slowly convalesces. The stools following these attacks are putrid, and in young infants are sometimes oily in character. The interval between the attacks may be as short as one week, or months may intervene. In the less severe forms of lithæmia the infant or child may be quite well during this interval, but, unfortunately, this is not always so. Some of these lithæmic children remain pale and frail-looking at all times. They are peevish and hard to please; they are as relentless as they are exacting in their demands. Lithæmic infants and children are mentally precocious, and when ill and peevish between the acute attacks they exercise this precocity in devising ways and means to secure the constant attention of all around them.

From the gastro-enteric type of lithæmia above described there are many variations. In children these attacks may occur, as they commonly do in adults, with little or no elevation of temperature. They may or may not be accompanied by convulsions, headache, gastric pain, or dyspnœa. The dyspnœa when it does occur is an interesting symptom, since it is not due to pulmonary causes, but is, like all the other symptoms, toxic in origin and to be classed as a nervous symptom. In rare instances vomiting of blood may occur both in the child and the adult, but this symptom does not change the prognosis or delay the return of the digestive organs to their normal condition. It is of importance in that such a lithæmic attack might be mistaken for gastric ulcer.

In infancy, childhood, and adult life a chronic intestinal fermentation may be dependent upon a lithæmic condition, but in these cases the symptoms which are always present as a result of chronic intestinal fermentation are at times aggravated into more acute attacks of gastro-intestinal disturbance. These acute gastro-intestinal attacks recur without apparent cause and at more or less regular intervals, in that way breaking in upon the milder gastro-enteric symptoms, which are constantly present. This type of lithæmia is, in the adult, commonly associated with great mental depression. It may also here be noted that the pain from these gastric attacks is not uncommonly so severe in the adult as to demand for its relief the hypodermic use of morphine. The lithæmic attacks of infancy and childhood are, fortunately, not so painful as they may be in later life. The gastro-enteric symptoms of lithæmia at all ages may vary in severity from a slight nervous dyspepsia to an attack of pain and vomiting so severe as not only to strike down, but even to endanger the life of, the patient.

Nervous Symptoms.—Nervousness in a great variety of manifestations is to be observed in lithæmic individuals. It might almost be said that the entire symptomatology of lithæmia at all ages may be directly or indirectly referred to the nervous system. Infants and children with strong inborn lithæmic

tendencies have very unstable nervous systems. The increased reflex excitability of these children predisposes them to general nervous irritability. They are commonly quick-witted, bright-faced, small and slender of stature, and flit about with quick and nervous movement. But lithæmic, unlike tuberculous, precocity is not, as a rule, coupled with physical inferiority; neither is lithæmic precocity so fitful, so asymmetrical, and so short-lived as the tuberculous. Lithæmic children, in fact, are, under proper restraint, capable of the highest intellectual development in after-life.

Eclampsia may be a symptom of lithæmia. In this connection the following abstract of a case reported by Irving Snow to the American Pediatric Society in 1893 is of interest. This case was reported under the title "Gastric Neurosis in Childhood," and the clinical history of this child conforms in almost every particular to the gastro-intestinal form of lithæmia above described. The lithæmic attacks from which this child suffered commenced when it was nineteen months old. The most characteristic symptom of these attacks was the initial *convulsion*. This was followed by from three to five days of fever and vomiting, and then rapid convalescence supervened. These spells were periodic; they came and went without apparent cause at intervals of a few weeks. Convulsions continued to mark the onset of the attacks until the child was four years old, when the convulsions ceased, but otherwise the attacks were unchanged, except that they were more frequent and possibly more severe. After the cessation of the convulsions the attacks were characterized by "vomiting, fever, hypersecretion, and irritability of the stomach, which were independent of dietetic errors or of organic disease." Following the report of this case, similar cases were reported by Holt, Christopher, Rotch, Seibert, Forchheimer, and Caillé, and the opinion was a common one that these cases were very frequently observed in practice, but that their etiology was obscure and their classification uncertain. I have here introduced the abstract of this case and the discussion which followed for the purpose of emphasizing the fact that eclampsia is not uncommonly associated with other well-marked lithæmic symptoms. I desire to emphasize this clinical relationship, since my laboratory experiments have demonstrated that eclampsia may be a symptom of lithæmia. The fact of greatest importance pertaining to lithæmic eclampsia is that these convulsions may continue to recur till finally we may have established the type of epilepsy which has been described as migrainous epilepsy.

Migraine is one of the most common, as well as one of the most characteristic, symptoms of lithæmia in adult life, and it is but slightly less important as a manifestation of this condition in childhood. These paroxysmal and commonly unilateral headaches occur at more or less regular intervals without apparent cause; they are sometimes associated with nausea, vomiting, and gastric pain, and not infrequently with disorders of vision. They are self-limited, and, as a rule, end in narcotism, which produces a sleep from which the patient awakens convalescent from the attack. Migraine is quite common in late childhood, and may occur in very young children. These lithæmic headaches may present two distinct clinical types: one that is associated with nausea and vomiting, and commonly called "sick headache;" and the other, in which there is not the slightest trace of these symptoms, may be designated as migrainous neuralgia. These clinical types of migraine are important from a therapeutic standpoint, since they do not yield alike to the same line of treatment.

In concluding the nervous symptoms of lithæmia it may be broadly stated that headache, gastric pain, nausea, vomiting, eclampsia, and rapid breath-

ing (asthma) are lithæmic symptoms which may occur in paroxysms, and which may be commingled in varying degrees of intensity to make the clinical picture of an individual attack.

Eczema is one of the most common of lithæmic manifestations in infants and children. Special note should be made of the importance of this symptom, since the successful treatment of this form of eczema depends upon the recognition and treatment of the lithæmic element. Lithæmic eczema may occur in well-nourished children with a family history of lithæmia, and is to be carefully differentiated from tuberculous eczema, since the two types require radically different constitutional treatment.

Urine in Lithæmia.—The urine excreted during a lithæmic paroxysm is, as a rule, scant and unusually acid in reaction. It is highly colored, and the specific gravity is generally considerably increased: on standing it deposits a red sand of urates. In the urines passed immediately following lithæmic headache, lithæmic eclampsia, and certain other of the more severe forms of lithæmia the poisonous xanthin bodies, paraxanthin and heteroxanthin, may be found in enormous excess of the normal minute quantities of these substances present in the urine of non-lithæmic individuals. Special note should be made of the fact that albumin may occur in the urine during, and for some days after, a lithæmic attack, and then entirely disappear. This recurrent and transient albuminuria is not a very common symptom of lithæmia, but when it does occur it is a very characteristic and significant one. It is, in fact, a danger signal, which being interpreted means that most careful treatment must be begun and continued if the kidney is to be saved from irreparable damage.

Treatment.—The dietetic treatment of lithæmia is of the first importance in infancy, as it is at all periods of life. Mother's milk is an ideal food for lithæmic infants, but when it becomes necessary to supplement this food it is best to do so with cow's milk to which cereals have been added. I have been much impressed with the importance of adding barley- or rice-water to cow's milk as a food for these children. Jacobi for many years has enthusiastically advised that cow's milk as a food for infants should always be mixed with cereals, and it is my experience that this is of special importance to lithæmic infants. Beef-juice and meat soups and teas are at all times contraindicated. When the lithæmic infant becomes a child, the milk and cereals, including bread, should continue to occupy the most important place upon his bill of fare. Milk and cereals are, in fact, ideal foods for lithæmics of all ages. As the child develops, it becomes necessary to add eggs, fish, and poultry to his diet. These foods are very much to be preferred to butcher's meat as a means of furnishing proteid food to the rapidly developing lithæmic child. Butcher's meat may, however, be allowed in small quantities once a day to lithæmic children who lead an active out-door life. In advising as to the proscribed and prescribed proteid foods for lithæmic children it is well to keep in mind that the following foods are to be recommended in the order named: Milk, eggs, fish, oysters, poultry, game, and butcher's meat. At the beginning of this list we have the best, and at the end the worst, foods for lithæmics of all ages. Fresh fruits and fresh vegetables should enter largely into the diet of all lithæmic children, and these foods, together with milk, eggs, and cereals, should constitute the almost exclusive diet until they are old enough to live a very active out-door life. Then, as above indicated, fish, poultry, and in small quantities butcher's meat, may be added. In the treatment of adults I advise that they eat moderately of simple food and abstain absolutely from wine and malt liquors. In this bit of

advice we have a condensed statement of the dietetic management of lithæmia. Over-eating is a factor in its cause, and under-eating is a factor in its cure. Lithæmics for this reason should be advised against taking an excess of food of any kind. Meats may be taken only in such quantities as are necessary to supply the proteid waste and repair of the body, but it will be found that most lithæmics take meat largely in excess of this quantity. It will therefore be necessary to place restrictions on the quantity of meat taken, and substitute poultry, game, fish, oysters, eggs, as above directed. No harm, however, can come to lithæmics leading an active life from the moderate use of these simple proteid foods. The only care necessary is to avoid an excess of these foods and to see that they are prepared in a simple and digestible form. Fries and salads are objectionable, and fresh pork, lobsters, and crabs are not to be commended. Sweets, such as candies, pastries, and preserves, are to be used sparingly if at all. The knowledge that sweets are injurious to lithæmics is a bit of information, born of clinical experience, upon which almost all writers are agreed. Sweets are therefore to be restricted, even though we cannot trace the connection between this class of foods and the nitrogenous poisons which are thought to be the cause of the symptoms of lithæmia. Milk, cereals, fresh fruit, and fresh vegetables should continue to be the most important foods of lithæmics throughout life.

Exercise in the open air is scarcely less important than diet to lithæmic children. They should, therefore, be encouraged in all kinds of out-door athletic sports. It will be found that many of these lithæmic children require a great deal of urging and commanding in order to have them take the proper amount of exercise in the open air. It is a common observation that lithæmic children are averse to out-door exercise and very fond of in-door intellectual pursuits. The out-of-school companions of lithæmic children should be bicycles, skates, and tennis racquets instead of books. Wholesome exercise in the open air is necessary to the proper physical and intellectual development of any child, but lack of exercise is especially baneful to one of inborn lithæmic tendencies. Exercise promotes the nitrogenous metabolism; it furnishes the conditions for the more complete oxidation of the alloxuric bodies into harmless nitrogenous extractives. The air in which the exercise is taken should be as pure as possible. City children of this type should have two or three months of active out-door life in the country every year. They may be sent to the seashore, the mountains, or a neighboring farm with almost equal advantage. Fothergill believed that a certain amount of pure country air was absolutely necessary to the satisfactory development of lithæmic children.

Before beginning the medical treatment of lithæmia one should make a careful search for such reflex factors as may possibly contribute toward precipitating lithæmic paroxysms. If eye-strain exists, it should be corrected. If pelvic or rectal disease be present, it should be treated. In short, all reflex factors should, if possible, be removed before other treatment is commenced. While I am convinced that the reflex factors have had undue prominence given them in the study and treatment of lithæmic paroxysms, yet I am not pessimistic enough to believe that they should be disregarded in the treatment of these conditions. Pelvic disease, I think, especially demands treatment when it occurs in cases where the lithæmic paroxysms coincide with the menstrual period. The failure of medicinal and dietetic treatment to cure certain lithæmic paroxysms may sometimes be due to the fact that there is present some eye, preputial, or pelvic disease which continues to act as a potent reflex factor in calling forth these paroxysms.

The medicinal treatment of lithæmia should aim to cure constipation and to favor the elimination and promote the oxidation of the alloxuric bodies which are believed to be the *materies morbi* of this affection. In infants and children it may advantageously be begun with small doses of calomel and soda repeated at short intervals until catharsis begins. After a day or two of rest from medication our little patients may be given some form of eliminative treatment. Volumes have been written on the drugs which are given for the purpose of eliminating the poisons of lithæmia, and there always has been, and possibly will be for some time to come, much confusion as to their comparative value. It is my belief that the salts of salicylic acid are the most valuable eliminative medicines we have. After the preliminary calomel course it is my custom to order some salicylate, the one selected depending upon the age of the child and the nature of the symptoms. Salol is especially useful. I have seen lithæmic infants suffering from chronic intestinal fermentation with gastric crises very much benefited by one grain of this drug after each nursing. Other antiseptics will not accomplish the same result, and it is not, therefore, simply a question of intestinal antiseptics. The salol in these cases must be continued for weeks or months in doses to suit the age of the child. If the lithæmic manifestation be an eczema, salol is equally advantageous; in such cases I also commonly give a few grains of phosphate of sodium or benzoate of lithium dissolved in each portion of food. An infant two years of age may be given in this way twenty grains of the phosphate of sodium and three grains of the benzoate of lithium in twenty-four hours. In a word, salol, phosphate of sodium, and benzoate of lithium are the medicines usually relied upon in the treatment of infantile lithæmia, and great good can be accomplished by their intelligent use in connection with such dietetic, hygienic, or local treatment as the special manifestations suggest. Should the phosphate of sodium fail to regulate the bowels (almost all of these cases are constipated), it becomes absolutely necessary to supplement this treatment with a laxative which will evacuate the upper intestine. Enemas and suppositories may be used as assistants to other laxatives, but they are not to be relied upon exclusively. I wish here to especially insist that this laxative treatment is as absolutely necessary in the lithæmia of infants and children as it is in adults. Salicylate of sodium may be advantageously substituted for salol in children over five or six years of age. The salicylate of sodium derived from winter-green is preferable, because it is more palatable and less irritating to the gastric mucous membrane. It should, if possible, be given in a little Seltzer water, which may for convenience be obtained in siphon. The siphon of Seltzer should be kept in a cool place, and the water may be drawn into a glass containing the dose of salicylate. In this way it is possible to give the drug for an indefinite time without disgusting the palate or irritating the stomach.

While the salicylates are our best remedies in all forms of lithæmia, the salts of lithium are also of value in certain manifestations of the disease. The natural lithia waters may be used, and it is much in their favor that these waters are tasteless, and therefore readily taken by infants and children. Much of their efficacy, however, is due to the water itself rather than to the lithia it contains. Many lithæmic patients drink little, and will be greatly benefited by simply increasing the quantity of liquid taken in twenty-four hours. Mention has previously been made of the importance of giving newly-born infants water to drink, since it is often needed to dissolve and thereby favor the excretion of urates that might otherwise irritate the inflamed urinary passages. For the same reasons lithæmic patients of all ages are benefited by drinking water, and much of the benefit derived

from drinking sulphur and other waters at the springs comes from the large quantity of liquid taken, rather than the contained medicinal agent. Yet in giving full credit to water as a remedy one must not overlook the fact that many natural waters contain salts—lithia, for example—that are of real value in the treatment of lithæmia. Of the lithia salts, the benzoate and citrate are much to be preferred, and I would select the benzoate, as it gives the best results. For infants the dose is gr. ss-j three times a day dissolved in milk; to older children it may be given in tablet form or dissolved in water. The citrate of lithium is somewhat less efficient, but more palatable, than the benzoate.

The soda salts are of great value in the treatment of lithæmia, and the mineral waters which are composed largely of these salts—such, for example, as Carlsbad—have a well-deserved reputation. The following prescription has long been a favorite with me for older children and adults:

R_x. Sodii salicylatis (from wintergreen) ʒij;
 Sodii phosphat., dry ʒiv;
 Sodii sulphat., dry ʒiiss.—M.

Sig. A teaspoonful, more or less, in a small glass of Seltzer water before breakfast every morning or every second morning.

It is important that dry salts be used in this prescription. The dose is to be regulated by the cathartic effect. Violent daily catharsis is not to be desired, but a decided laxative effect must be produced. In connection with this treatment I commonly use one of the following prescriptions:

First: A one-grain salol-coated pill of permanganate of potash (Upjohn), which is to be given directly after each meal to all lithæmic patients having pronounced gastro-intestinal symptoms. (Sick headache and the gastro-enteric types of lithæmia belong to this class.)

Second: A capsule containing from two and a half to five grains of salol and from one-twelfth to one-quarter grain of cannabis Indica, which is to be given after each meal to all patients in whom the lithæmic paroxysms are not associated with gastro-enteric symptoms. (Migrainous neuralgia and lithæmic epilepsy belong to this class.)

These prescriptions are to be used in connection with the soda salts, and are especially adapted for the treatment of lithæmia in late childhood and adult life. They are not suited to young children or to frail and wasted lithæmics of any age.

Dilute nitro-muriatic acid and colchicum have long held a place among medicines which are of value in the treatment of lithæmia. Both may be given to older children and adults, but are not to be employed in infants and young children. The dilute nitro-muriatic acid in five-drop doses, well diluted, before meals, is a valuable remedy in the treatment of lithæmic headaches in older children. The wine of colchicum in five- to eight-drop doses may be tried for the relief of painful lithæmic paroxysms of any kind.

For stout and vigorous patients the natural waters are of great value, especially those of the thermo-alkaline springs of Virginia and Arkansas and waters of the Carlsbad type. The Bedford Springs of Pennsylvania, the Crab Orchard Springs of Kentucky, the St. Clair and Mount Clemens Springs of Michigan, the Saratoga Springs of New York, and the West Baden and French Lick Springs of Indiana may also be recommended.

HEREDITARY SYPHILIS.

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No period of life is exempt from syphilis, which has been aptly styled "the least venereal of the venereal diseases." It is a chronic infectious process, doubtless of microbic origin, the ravages of which are modified by age, conditions of body, and environment. The micro-organism most commonly associated with syphilis as a probable causative agent has been found by Lustgarten within the cellular protoplasm of syphilitic products. He describes it as a bacillus from three to seven micro-millimetres in length, with often a slightly wavy shape. Unfortunately, pure cultures have not been made of this bacillus, and the fact that the lower animals do not contract syphilis prevents the possibility of proof by inoculation.

Syphilis in early life may be either hereditary or acquired. It is not necessary to consider acquired syphilis at length in a work devoted to diseases of children, as it presents no essential differences from the same affection in adult life. It may be well to bear in mind, however, that syphilis detected in infancy is not necessarily inherited, but may be acquired. A primary sore upon the genital tract of the mother may infect the infant during birth, though the possibility of this has been denied. The nurse or attendant may have a primary lesion upon breast or lips. Much more common will be infection from some secondary lesion, especially a mucous patch upon the mouth or lips. There are many ways in which the blood or infective secretions of a syphilitic patient may come in contact with a solution of continuity in the skin or mucous membranes of an infant or child. In such a case a chancre will appear at the point of contact, followed in due time by the after-lesions of the disease. There are certain peculiarities in the effect of the syphilitic virus upon young protoplasm which will be noted under the Morbid Anatomy.

The subject will be here considered under the two heads of hereditary syphilis in infancy, and the taint as it is seen in childhood or when apparently delayed.

HEREDITARY SYPHILIS IN INFANCY.

The disease may be acquired from the father or mother, or from both parents, the poison being lodged in the spermatozoa of the male or the ovum of the female.

Paternal Influence.—While it has been denied by some observers that the father alone can transmit syphilis, the consensus of opinion is in favor of the possibility of such transmission, which can and does take place. The chances of this transmission depend upon certain factors, such as the stage of the disease and the degree of its intensity, as well as the thoroughness with which treatment has been followed. Without mercurial treatment the sperma-

tozoa can usually transmit the syphilitic poison during the first year after primary infection, and there is great danger to the foetus from syphilitic contagion up to the fourth year. The longer the duration of the disease, the less will be the danger to the offspring, owing to the periods of latency observed during its later stages. If the father be subjected to early and thorough treatment, the probability of transmission of the disease will be much lessened, and such a possibility soon becomes lost with a reasonable lapse of time. If the father infect the mother, as frequently happens, there will be a double syphilization of the offspring, which will probably be stillborn or soon succumb to an aggravated form of the disease.¹

Maternal Influence.—The influence of the mother upon the growth and development of the foetus contained within her uterus is obviously very great, and hence when she is suffering from constitutional syphilis the disease is transmitted in an active stage to her child. The degree of such transmission depends, as noted above in the case of the father, upon the stage and severity of the disease and the nature of the treatment employed. During periods of latency the mother may bear healthy children, followed by abortions or syphilitic infants caused by renewed manifestations of the disease. It has been considered that the power of transmission is practically lost at the end of six years. As a general rule, it can be stated that the chances of infection of the foetus and the severity of the type, if infected, are in direct proportion to the activity of the syphilis in either or both parents. It has been said that if the mother contract syphilis before the eighth month of utero-gestation, she may transmit the disease to the foetus, although healthy at the time of conception. Dr. Taylor, on the contrary, denies that the syphilis of the mother, acquired during pregnancy, can be conveyed to the foetus through the utero-placental circulation, as the disease is only communicated either by the sperm-cells or by the ovule diseased at the time of conception. One of the peculiar phenomena seen in connection with infants who are born syphilitic is that the mother may apparently be free from any taint of the disease. It has been a subject of much dispute whether these are instances of latent syphilis or whether the women are really healthy. Whatever the cause, these cases show immunity in contracting syphilis.

In 1837, Colles wrote that "a new-born child affected with inherited syphilis, even although it may have symptoms in the mouth, never causes ulceration of the breast which it sucks if it be the mother who suckles it, although continuing capable of infecting a strange nurse." The substantial truth of this dictum has not been seriously questioned during the many years that have elapsed since its enunciation, although varying explanations have been offered. Fournier states that the inoculation experiments of Caspari and Neumann have proved conclusively that the apparent immunity of the mother, who has borne a child syphilitic by its father, against the contraction of the disease from her offspring, is due to the fact that she has already been infected by syphilis during the intra-uterine period of the child's life. Thus, conceptional syphilis is to be classed with the hereditary form of the disease, since there is here no primary lesion. This form of conceptional syphilis may remain latent for years. Diday advances as an explanation of Colles' law the idea that all infectious diseases may certainly be mitigated to the point of absolute protection by the methodically repeated inoculation of their essential cause (microbic) or of its products (toxic ptomaines, etc.). Bouchard considers that while the foetus retains the supposed pathogenic agent itself, the products dissolved in the blood find their way to the tissues of the mother and set up a nutritive change,

¹ Dr. F. R. Sturgis strongly denies the paternal transmission of syphilis.

resulting in what he calls a "bactericidal condition," which renders difficult or impossible the development of the infectious agent when introduced by later inoculation, as from the lips of her child. The doctrine of syphilis being contracted by conception, sometimes called "choc en retour," although having wide acceptance, is not acknowledged by all. Kassowitz believes that the women who appear healthy and remain so, even after giving birth to syphilitic children, are really free from specific taint.

SYPHILIS OF THE PLACENTA.—Dr. Fränkel in 1873 published a paper in which he affirmed the existence of three forms of involvement of the placenta by syphilis—*i. e.*, endometritis decidualis, endometritis placentaris, and disease of the villous portion of the foetal placenta. This conclusion was based upon an examination of over one hundred placentæ. Zilles in 1885 published the results of a study of three hundred placentæ derived from Prof. Säxinger's obstetrical clinic. He finds that placental syphilis can often be diagnosed microscopically, and that it oftenest happens in connection with foetal syphilis. The maternal portion of the placenta or the foetal part only may be affected, while, again, the whole of the placenta may be involved in the disease. Syphilis is one of the recognized causes of hydramnios.

Morbid Anatomy.—The lesions of syphilis, while always essentially the same, will nevertheless be modified by age. Young protoplasm is active, and usually exhibits a marked reaction to irritative processes, so that the tissues are apt to be extensively involved in hereditary syphilis. The lesions may be broadly divided into those involving the skin and mucous membranes, the viscera, and the bones.

SKIN AND MUCOUS MEMBRANES.—The skin may be affected by erythema, maculo-papules, or papules. A vesicular and pustular eruption may occasionally be seen. Blebs or bullæ often appear at birth in a severe type of the disease. Crops of boils, with well-defined, coppery-red bases, are apt to be symmetrically arranged when many are present, or asymmetrically distributed if only a few are seen. The distribution and course of the various eruptions will be noted more at length under Symptoms. In general, they develop quickly and spread over extensive areas of surface on account of the character of infant protoplasm, noted above, as well as from the activity of the circulation in the skin.

The lesions of the mucous membranes may be in the form of catarrhal processes, of mucous patches, or of superficial or deep ulcerations. Any or all of these lesions may involve any part of the alimentary tract or of the respiratory tract. They are seen most commonly, however, in the upper part of these areas, in some part of the mouth or fauces in the former case, and in the nose and larynx in the latter. Still, they may likewise occasionally involve the intestine or trachea and bronchial tubes.

VISCERAL LESIONS.—The viscera are apt to be more extensively involved in hereditary than in acquired syphilis, the lesion being in the form of an interstitial hyperplasia more or less diffuse. Circumscribed gummy infiltrations are not so frequent. The growth of interstitial connective tissue, which gradually contracts, thereby partially obliterating the parenchyma of the organ, may involve the lungs, spleen, liver, pancreas, and testicle.

Lungs.—Usually a portion of a lobe, but occasionally a whole lobe, may present a diffuse fibroid infiltration. The part involved is grayish-white in color and tough in consistency, and surrounded by an inflamed pleura. Under the microscope there is seen to be thickening of the septa and compression of the alveoli by fibrous tissue, which is quite vascular. Occasionally a few rounded masses about the size of a hickory-nut may be noted. These gum-

mata may break down in the centre into puriform matter, but they are not apt to exist in the same subject that the diffuse interstitial inflammation attacks.

Spleen.—The spleen is generally more or less enlarged from a diffuse interstitial hyperplasia. There usually coexists a thickening of the capsule. According to Dr. Gee, the severer the grade of syphilis the greater will be the hypertrophy of the spleen. This enlargement may remain persistent for a long time after other symptoms have disappeared.

Liver.—The liver, which is not infrequently affected, is hardened and enlarged from a diffused sclerosis. Occasionally the affection may be circumscribed. The hepatic cells are compressed and the capillary blood-vessels partly obliterated by the pressure. As in cirrhosis in the adult, section of the liver is accompanied by creaking, and the cut surface presents a yellowish area, interwoven with whitish opaque streaks of fibro-plastic matter. The capsule of Glisson may be thickened upon the surface of the liver, and there may be local peritonitis. Gummata, in the form of small, circumscribed nodules, may be found in the tissue of the liver. They may be seen in association with cirrhosis. These nodules are yellowish, with a tendency to soften in the centre.

Pancreas.—Birch-Hirschfeld has called attention to the fact that there may be hyperplasia of the connective tissue of the pancreas, which on section presents the same fibroid appearance seen in the liver and other visceral organs thus affected. He found in a few cases the head of the organ more involved than the remaining part of the gland.

Testicles.—An interstitial orchitis may affect one or both testicles, producing hardening and slight enlargement of the glands. The hyperplasia may be uniformly distributed through the organ, or the latter may be irregularly involved. The epididymis is not usually affected. Atrophy of the seminal ducts may ensue. Sufficient change in the testicle to be detected clinically is not often seen in hereditary syphilis.

Kidneys.—Parrot has found small tumors, produced by infiltrations of round cells into the connective-tissue stroma, which compress the tubules, and thus cause a colloid degeneration of the contained epithelium. If this process is extensive, it will eventuate in a general atrophy of the kidney. General nephritis may be seen in hereditary syphilis, but it is difficult to say whether the latter is more than a predisposing cause of the former condition.

Heart.—Gummata may be found in the heart. Dr. Coupland has reported a case where the walls of this organ were thickened and hardened.

BONE LESIONS.—Waldemeyer, Köbner, Parrot, and R. W. Taylor have shown that various bony lesions are quite common in hereditary syphilis. Many of these lesions, that were formerly referred to rickets or scrofula, are now recognized as syphilitic. There are two principal ways in which the specific poison affects the bones in early life. In one instance the brunt of the disease and morbid change takes place at the junction of the shaft with the epiphysis; in the other, the periosteum covering the long bones is principally affected. Both of these varieties involve principally the long bones.

Osteo-chondritis.—This inflammatory process is induced only by syphilis, and may be the sole manifestation of the taint. The lesion starts in the cartilage joining the epiphysis with the diaphysis, where normal growth in length of the bones takes place; hence deformity of the bone, due to a crippling of its proper development, may ensue. The lesion most commonly affects the bones of the forearm, leg, arm, and thigh, although other bones may be involved, such as the metacarpal and metatarsal bones, the clavicle, sternum, and ribs.

The number of the bones affected appears to depend, to a certain extent,

upon the severity of the general poisoning. It has been found in stillborn infants that most of the long bones may be thus affected, and in those born living, if the bone lesion is multiple, recovery is uncommon. The cartilage affected first becomes thickened and soft from proliferation of cartilage-cells, and there is at the same time lessening of the intercellular substance. This may be felt as a sort of collar-like swelling at the end of the bone affected. The swelling may be visible if the child is not too fat. If, as occasionally happens, one portion or side of the cartilage only is involved, the swelling will be felt not to completely encircle the bone, but as a circumscribed nodule. The disease is apt to be symmetrical and involve the distal oftener than the proximal ends of the bones. There is little change in the integument or surrounding tissues in many cases, as the disease is not apt to extend farther than the bone. In such a case the swelling may remain for a long time, accompanied by little pain or disability. It may originally develop slowly or quickly, and its disappearance will usually promptly follow a proper mercurial treatment. In some cases, however, degenerative changes may ensue, with a breaking down of some part of the swelling. If the morbid process continues, there will be softening, soon followed by ulceration of the skin. If suppuration keeps up, the cartilage will be destroyed and the epiphysis completely separated from the diaphysis. Even in these cases the joint is not apt to be involved, although cases of subacute synovitis, and even pus in the joint, have been reported. If the ulceration is extensive, the epiphysis, when completely separated, may be extruded. When there is destruction of the cartilage and epiphysis, there will of course ensue arrest of growth and consequent deformity in the limb. Parrot has described cases in which the skin remains unbroken after separation of the epiphysis, inducing a condition of paralysis in the affected part. Dr. Taylor describes cases in which, the intervening cartilage having been destroyed, the epiphysis is united to the shaft only by fibres of periosteum. This membrane may become much thickened, and form a more or less complete cylinder, uniting the two fragments with considerable firmness. Bony spiculæ shoot from its inner surface between the two osseous surfaces, and thus eventually bony union is secured. The swollen periosteum may gradually resume a more nearly normal thickness.

Osteo-chondritis develops early in life, usually within the first month. The lesion may, however, occur later, when it is not apt to become multiple, and may be unsymmetrical in distribution. The question as to whether certain epiphyseal swellings may be due to syphilis or rickets will possibly arise. Other lesions of these two diseases will have to be sought after in order to aid in making a correct diagnosis. Such swellings are pretty surely syphilitic if they occur during the first six months of life, and at all times are relieved by mercurial treatment. Again, the epiphyseal swellings of rickets are always symmetrical, while those of syphilis may be unilateral.

Periostitis.—This form of lesion occurs later in hereditary syphilis, usually after the child has begun to walk. It attacks by preference the femur, tibia, and bones of the forearm, occurring usually from the second to the fourth or fifth year. There is more or less enlargement of the affected bone. At an early stage of the disease the bones are attacked symmetrically, but later circumscribed nodes may be placed unilaterally.

Dactylitis.—The phalanges and the metacarpal and metatarsal bones may be enlarged to several times their natural size. After an interval of time the skin may become inflamed and break down from the formation of an abscess. The proximal phalanges are more apt to be attacked than the distal, and several bones of each hand may be affected. There is not much destruction of

bone, even in severe cases, and, although the disease tends to run a slow course, it is always influenced favorably by treatment. Dactylitis is apt to occur in very young subjects, when it takes the form of a gummatous infiltration. (Fig. 1).

FIG. 1.



Syphilitic Dactylitis.

Craniotabes.—The local thinning of portions of the cranial bones was formerly attributed exclusively to rickets, but is now known to ensue as well in the malnutrition accompanying syphilis. As it is due to pressure of the thin skull between the brain and pillow, it is especially apt to involve the occipital bone. Carpenter considers that both craniotabes and Parrot's nodes are often syphilitic manifestations, although they are more frequently regarded as evidences of rickets: 74 per cent. of cases of craniotabes are syphilitic, according to this author.

Symptoms.—The symptoms of hereditary syphilis vary widely according to the extent of the poison. When the virus is concentrated, as in cases where both parents are syphilitic, the foetus will be attacked by the disease in the uterus, and, as a result, we shall have abortion more or less early in the pregnancy. As the disease abates in one or both parents the pregnancies will be longer in duration, until finally apparently healthy infants may be born.

In some cases the infant will present marked evidences of syphilis at birth; often, however, the onset is delayed until later, and at birth there may be absolutely no manifestation of the disease. In 158 cases analyzed by Diday the first manifestation of symptoms occurred in 86 cases before the completion of one month; in 45 before the completion of two months; and in 15 before the completion of three months after birth. The remaining 12 cases showed the symptoms in intervals varying from four months to two years.

The earlier the disease manifests itself after birth, the graver will be the nature of the attack. Very early syphilis is usually accompanied by emacia-

tion, eruptions of bullæ, particularly upon the palms of the hands and soles of the feet, and an extreme degree of coryza, cracked and ulcerated lips, and evidences of visceral and bony disease. In the older cases there may be no interference with nutrition, and possibly one or two mucous patches may be the only active manifestation of the disease. In studying the symptoms it may be well to consider the disease as it shows itself in different structures and areas of the body.

Skin.—One of the early symptoms appearing upon the skin will be the eruption of small round pink spots, disappearing on pressure, and usually appearing first on the lower portion of the abdomen. It may spread from this location and finally involve the whole body. Pigmentation of these spots may ensue, and they may present a dark-red, coppery discoloration. This latter change may be considered as having a diagnostic value. In hereditary syphilis the rashes often develop rapidly, and are apt to be less symmetrical than those seen in adults. They are likewise polymorphous, as several different forms of syphilide may be exhibited at the same time in a given case. A papular syphilide may be seen in the form of small or large flat papules, symmetrically distributed over the surface. These papules are not so apt to group themselves into lines and circles as in older subjects with syphilis. They are not so solid and deeply infiltrated as in the adult. Upon the palms and soles these papules may be very abundant and fuse together, presenting a thickened, dull-red surface. The vesicular syphilide is not common, and when seen is apt to be in very severe cases. The vesicles may be associated with pustules, and appear in closely-arranged groups about the mouth or chin or various other parts of the body, especially the nates and hypogastrium. Pustules may form, especially on the face, buttocks, and thighs. The ulceration is deeper and the crusts darker in color than in impetiginous eczema. Pemphigus likewise appears in the severe forms of the disease. It most frequently attacks the palms of the hands and soles of the feet; it may have a copper-colored areola and develop rapidly. Crops of indolent boils, symmetrically distributed and of a copper-red color, may appear in connection with other eruptions. They are more apt to be seen in badly-nourished infants. In some cases the only appearance of syphilis upon the skin will be a smoky discoloration, seen most distinctly in the prominent parts of the face, such as the eyebrows, cheek-bones, and bridge of the nose. The nutrition of the skin is much affected in cases where the cachexia is marked; it hangs in dry, loose folds, having an unhealthy, earthy appearance.

Mucous Membranes.—The mucous membranes, as well as the skin, present the earliest manifestations of the disease. One of the most typical lesions is the coryza, which may be the first symptom noted. First, there may be a serous discharge which attracts little notice; this, however, gradually becomes worse, and the nasal secretion takes on a purulent or even a bloody character, and may be sufficiently irritating to cause excoriations of the upper lip. The mucous membrane itself becomes thickened, and the inspissated secretion soon dries, forming crusts, which may completely block up the passage through the nostrils and seriously interfere with nursing. The secretion may likewise be offensive. In severe cases, particularly where cleanliness is not practised and the decomposing secretions are allowed to remain in the nostril, there may follow ulceration of the mucous membrane, and possibly even necrosis of the adjacent bony parts. There is apt to be a flattening of the bridge of the nose, probably, to a certain extent, due to the interference with normal respiration. The inflammation may spread to the pharynx and larynx, although its action is likely to be limited to the Schneiderian membrane.

Mucous patches will be seen in most cases of hereditary syphilis, and, although they appear most constantly on the mucous membranes, they may be present upon the skin, particularly at its junction with the mucous membranes, or upon those parts which are thin and exposed to various secretions. They may occasionally be seen on any part of the cutaneous surface of the body. They are oftenest seen in the mouth, about the nose, upon the scrotum, vulva, labial commissures, and occasionally at the umbilicus. In the mouth the most frequent situations are upon the angles of the lips, inside of the cheeks, the pillars of the fauces, the tonsils, and the sides and dorsum of the tongue. They consist, in the early stage, of a slightly raised segment of mucous membrane, presenting a whitish surface and red margins. This may soon ulcerate. When the mucous patches appear at the angles of the mouth, deep fissures will often form at the corners of the lips, extending sometimes well out into the cheek. These fissures are sometimes called rhagades, and are diagnostic. The secretions on these mucous patches are very infective. When mucous patches appear on the cutaneous surface, they are slightly raised, with a macerated appearance, and frequently seamed with erosions or cracks. In the late stages of hereditary syphilis mucous patches are not so numerous as in the earlier stages of the disease, but they frequently recur after the child is apparently restored to health.

Disturbance of Nutrition.—The extent to which the general nutrition of the infant is disturbed will depend upon the severity of the attack. In grave cases there is atrophy of all the structures of the body, the infant presenting a weazen appearance, with a countenance resembling that of an old man. These cases are almost invariably fatal, and are caused by the blighting influence of the virus. In many cases, however, a failure of nutrition will ensue gradually, consecutive to gastro-intestinal disturbance. This may be due to actual specific disease of the liver, stomach, or intestines, or it may be due to indigestion and malassimilation only indirectly caused by feebleness from the cachexia. In bottle-fed babies digestive disturbances are marked and severe, infants upon the breast being much less liable to suffer. In some cases the infant will present very slight disturbance of the general nutrition, being plump and well-nourished throughout the course of the disease, which may be only manifested by mucous patches or mild evidences of the infection.

Condition of the Blood.—A condition of profound anæmia is frequently seen, particularly in severe cases. Johann Loos states that hereditary syphilis is always associated with an anæmia which under some conditions may reach an extreme degree of intensity. This anæmia is characterized by a diminution in the number of the red blood-corpuscles, by quite a marked alteration in these corpuscles, the appearance of megalocytes and microcytes, and by the appearance of nucleated erythrocytes, sometimes in quite notable quantity. It is always characterized by the constant existence of leucocytosis, which may often become extreme, and by the appearance of myelo-plaques in the blood. This anæmia is a very important and significant symptom of the disease, and may directly occasion a fatal issue. He further states that there are only two diseases common to childhood in which the lesions of the blood suggest the changes just described, and these are splenic anæmia and severe forms of rachitis.

A form of syphilis hæmorrhagica neonatorum has been described by Bumstead and Taylor. There may be simply a limited subcutaneous effusion, or the mucous membranes may be the seat of the hæmorrhage. Hæmorrhage at the umbilicus shortly after birth may be due to this cause.

Glandular Enlargements.—General adenopathy is not seen in the hereditary

form of syphilis. There may be enlargement of the chains of cervical glands consecutive to lesions in the adjacent mucous membranes, and occasionally there may be an affection of the inguinal, axillary, or cervico-maxillary glands without any deeper lesions being noted to account for their existence by septic absorption. The glands are hard, moving without pain in the areolar tissue under pressure by the finger. Some writers consider that enlargement of the epitrochlear glands is pathognomonic of congenital syphilis, but well-marked cases occasionally fail to show this sign upon careful examination.

Bony Organs.—The frequency with which the bones are involved in hereditary syphilis has been noted in the morbid anatomy of the disease. In every case the long bones should be carefully examined for enlargement and thickening at the epiphyseal and distal ends. In cases where suppuration has taken place the epiphysis may be separated from the shaft, and crepitation will then be found upon careful handling. The joint itself may occasionally be involved in the inflammation, showing the well-known symptoms of arthritis. Where the bones are much affected there will be some disability of the limb, possibly extending to complete paralysis. Immobility in such a case is without doubt due to the affection of the bones.

Dactylitis.—In the early period of the disease an enlargement of the phalanges is frequently seen, and occasionally also of the metatarsal and metacarpal bones. The proximal phalanx is more frequently attacked than the distal; the affection may spread to all of the phalanges, but is more apt to involve only one, which may be enlarged to double its normal size. This enlargement is the result of specific inflammation of the bone and periosteum, and runs a slow course unless modified by specific treatment. There is not apt to be much involvement of the soft parts; the integument will be reddish and inflamed, but there is little tendency to suppuration and ulceration. These swellings usually present a fusiform shape, with a hard, firm sensation to the touch.

Teeth.—The appearance of the deciduous teeth is delayed in hereditary syphilis, as in rachitis. The first teeth may not appear until the tenth or twelfth month, or even later. These teeth are poorly developed and apt to undergo early decay. There is usually a similar delay in the appearance of the second teeth, which present more pathognomonic changes, which will be noted in connection with late hereditary syphilis.

Nervous Disturbances.—Lesions of the nerve-centres do not often appear in hereditary syphilis; there may be, however, an occasional palsy due to a peripheral cause. One form in connection with bony lesions has already been mentioned. There may be contractures and paresis, however, where no bony lesion can be noted. Henoch questions whether such affections may not be myopathic in their origin and independent of the nervous system.

The following case coming under my observation illustrates a case of paralysis evidently caused by interstitial syphilitic myositis: An infant four weeks old, whose mother presented syphilitic lesions, was born healthy at full term. When seven days old it was noticed that the right leg was drawn up and apparently did not move; also the right arm. There was complete loss of power in these members; there was wrist-drop, and a loss of faradic and galvanic irritability in the extensors of the left wrist. The muscles affected were rather hard and painful to the touch. There was an enlargement at the epiphyseal end of the left humerus. The paralysis completely disappeared in about two months under specific treatment.

Dr. Eustace Smith states that a form of real paralysis has been occasionally seen affecting the branches of the brachial plexus, causing more or less complete loss of power in the arm.

Onychia.—Two kinds of onychia are noted in hereditary syphilis—the ulcerative and the nutritive. In the ulcerative form the pustule appears at the margin of the nail, which soon breaks down, leaving a sloughy surface, which may destroy the matrix. The surrounding skin presents a coppery discoloration. In the nutritive form, which is apt to appear later, the ulcer has a sloughy base, and presents a swelling around the periphery of the nail, which becomes thickened and brittle. Swelling and deformity of the phalanx may ensue. In a case recently observed, a child of two years, whose father had a specific history, presented immense bulbous masses upon the extremities of the thumb and middle finger of the right hand and the thumb and fore finger of the left hand. These were granular, warty masses about the size of hickory-nuts, with the nail protruding backward. When the infant was eight months old it appeared healthy, except that the finger-nails now involved were like claws and were reddened as if scalded. The trouble had continued until the nutritive changes produced the enlarged mass here noted. There had been a history of “snuffles,” abscesses on the buttocks, sore lips and gums, but at the time of the examination the only other manifestation of the disease was a large mucous patch in front of the scrotum. In the nutritive form of onychia the hyperæmia of the matrix and the deformity of the phalanx, if not extreme, may disappear under specific treatment.

Iritis.—This is an exceedingly rare affection in hereditary syphilis, but cases have been reported by Mr. Hutchinson in infants varying in age from six weeks to sixteen months. It does not differ from the same manifestation in adults.

Alopecia.—There may be loss of hair in the scalp, eyebrows, or eyelashes. The last form is the most pathognomonic, as there may be a deficiency in the nourishment of the hair of the scalp in rickets or any condition of cachexia in infants.

General Irritability.—Syphilitic infants are very fretful, and the cry is of a peculiar high-pitched character. This fretfulness is particularly apt to be present at night, at which time the child is extremely wakeful. In this, however, it does not differ much from rickets.

Diagnosis.—A difficulty in the diagnosis of hereditary syphilis may obtain where typical lesions are not well marked, or where it is a question between syphilis and scrofulous or tubercular lesions. In cases of marasmus, if there is no history of chronic indigestion, particularly if the infant have been fed at the breast, there is strong suspicion of syphilis. A careful examination for mucous patches will often throw light on such a case. Chronic coryza is likewise a valuable sign in diagnosis.

The following points of distinction between syphilitic and scrofulous lesions of the skin have been given by Dr. P. A. Morrow: (1) Syphilitic lesions are general in their distribution; they may occur upon any region of the body. Scrofulous lesions are more limited in their localization; they have a special predilection for the neck or regions rich in lymphatic glands. (2) Syphilitic lesions are ambulatory and changing; they disappear and reappear elsewhere. Scrofulous lesions are fixed and permanent. (3) The color of syphilitic lesions is reddish-brown or “lean-ham” tint. The color of scrofulous lesions is brighter and more violaceous in hue. (4) Syphilis is distinct from scrofula in its objective appearances and mode of evolution. In the initial stage the syphilitic neoplasms are firm and hard; the scrofulous infiltrations are softer and more compressible. In the ulcerative stage the differences are more pronounced; the ulcers of syphilis are cleaner cut, regular in contour, with perpendicular, firmly-infiltrated borders encircled by a pigmented areola;

scrofulous ulcers are irregular, with soft, undermined borders; they are painless, bleed easily, and show slight tendency to spread. (5) The crusts of syphilis are bulkier, thicker, with a tendency to accumulate in layers, and darker in color; the cicatrices are smooth and remain long surrounded by a pigmented areola. The crusts of scrofula are softer, more adherent; the cicatrices are elevated, irregular, bridled; they retain their violaceous color for a long time. (6) The course of a syphilitic ulcer, though sluggish and chronic, is much more rapid than that of scrofula. (7) Absence of pain and local reaction characterize both syphilitic and scrofulous ulcers; they are essentially lesions without symptoms.

In connection with the bony lesions it is important to diagnose between syphilis and tubercular and rachitic affections. The following points in diagnosis between syphilis and tuberculosis are given by Dr. Morrow: (1) Syphilis exhibits a marked predilection for the long bones; its habitual localization is in the diaphysis, and almost always at its terminal extremity. Tuberculosis is almost exclusively situated in the epiphyses, rarely affecting the shaft. (2) In syphilis there is a marked enlargement of the bone by more or less voluminous osseous tumors or hyperostoses, with little or no involvement of the soft parts; and in tuberculosis the tumefaction is due less to increase in the size of the bone than to œdematous infiltration of the soft structures. (3) In syphilis there is little tendency to suppuration and necrosis; in tuberculosis the pyogenic tendency is marked. (4) In syphilis osteocopic pains, with tendency to nocturnal exacerbation, are a pronounced feature; in tuberculosis the pain is dull and heavy, not aggravated at night; sometimes there is entire absence of acute painful symptoms. (5) The osseous lesions of syphilis rarely react upon the general system, while those of tuberculosis often determine a marked impairment of the general health, grave complications, hectic fever, cachexia, etc.

In syphilitic dactylitis there is little involvement of the soft parts, the swelling being caused by the enlargement in the size of the bone. In tubercular dactylitis the swelling is due more to an œdematous infiltrated condition of the soft tissues than to enlargement of the bone. In the latter cases breaking down of the tissues and ulceration are more apt to ensue.

The diagnosis between syphilis and rickety bone-lesions may be of great importance. Epiphyseal swellings occurring under six months are very apt to be syphilitic. In syphilis the epiphyseal swelling may be unilateral, but it is always symmetrical in rachitis. In doubtful cases the swelling must be subjected to specific treatment. It is well to remember, however, that rickets and syphilis may coexist in the same case. There is almost invariably enlargement at the costo-chondral articulations in all cases of rickets, which is absent in syphilis.

Prognosis.—According to Kassowitz, one-third of all syphilitic children die before their birth, and among those who are born 34 per cent. die in the first six months of life. Fournier places the mortality, when derived from the father alone, at 28 per cent.; from the mother alone, 60 per cent.; when from both parents, 68½ per cent. The earlier the symptoms appear after birth, the severer will be the type of the disease and the worse the prognosis. Involvement of the bones and viscera means a severe type of the disease. Infants fed upon the breast will have a much better chance than those artificially fed. In bottle-fed infants, particularly when the disease appears early, the prognosis is almost always fatal; it is invariably so in hospitals and lying-in institutions. Any interference with digestion and assimilation, no matter from what cause, will necessitate a guarded prognosis. If the coryza is extreme and breathing much disturbed, the prognosis must be altered in proportion to the amount of

such disturbance, which interferes with rest and the taking of food. If the digestion remains good, and particularly when the manifestations of the disease are not severe, complete recovery takes place, and the infant may grow up healthy and strong.

LATE HEREDITARY SYPHILIS.

In some cases of hereditary syphilis the manifestations of the disease during infancy may be exceedingly mild, and, in fact, overlooked. It is possible in such a case that the poison may show itself in various ways during the period of childhood. "*Syphilis tarda*" is a term applied to those cases in which the first manifestations of hereditary syphilis appear in childhood. The existence of such a condition without any earlier evidence of the disease has been disputed. It is analogous to the discussion as to whether syphilis in the adult may present late secondary or tertiary symptoms without being preceded by earlier lesions.

Late hereditary syphilis may manifest itself either in certain active lesions plainly to be attributed to this condition, or by certain developmental defects that may easily be confused with scrofula, tuberculosis, or rickets. It may be well for us to note some of the more characteristic lesions.

Bone Affections.—One of the commonest manifestations is a periostitis involving various long bones, especially the tibia, the ulna, the radius, and the humerus. Accompanying this periostitis there may be considerable thickening upon the surface of the bone, sufficient to induce a change in its form. The lesion may be multiple and symmetrical, although occasionally unilateral. It is attended often with little discomfort aside from occasional nocturnal pains. The nasal bones may be affected, producing much deformity by destruction of the bony arch of the nose. In many cases not so severe there is marked flattening of the bridge of the nose and a wide separation of the eyes. The frontal bone is apt to be large and flat, with prominences somewhat exaggerated. There is also usually a very high palate arch. Dactylitis may be seen in this late stage of the disease, and sluggish swellings of the metacarpal and metatarsal bones. The secondary teeth are affected in a way that has been considered pathognomonic. As is well known, Mr. Jonathan Hutchinson first called attention to this condition. The principal change is noted in the two superior middle incisors, which are small, peg-shaped, and placed at such an angle that the cutting borders, if continued, would meet. They may occasionally be deflected outward, as in the accompanying illustration. (Plate IV.) The cause of this maldevelopment has been explained by Fourrier as due to defective growth within the alveolus, while Hutchinson refers it rather to an early stomatitis or an alveolar periostitis often present during infancy. The incisors are apt to be notched at the lower edge, as is well shown in the plate, which is taken from a case under the care of Dr. Stowell. The enamel is usually eaten away in this portion of the teeth. Dr. John N. Mackenzie has called attention to ulceration of the palate, which is apt to take place in the centre, and be followed by caries or necrosis of the bone. There may be simultaneous or consecutive deep ulceration of the palate, pharynx, and naso-pharynx at any time previous to the age of puberty. Large and indolent mucous patches may be present upon the cheek, tongue, gums, and especially about the corners of the mouth. The ulceration about the lips may leave long scars, particularly to be seen at the commissures of the lips. This is most beautifully shown in the accompanying illustration of Dr. Stowell's case. (Plate V.)

PLATE IV.



HUTCHINSON TEETH.



FISTULÆ, OR RHAGADES.

(From Dr. Stowell's Case.)

Jacobi prefers calomel, on account of the rapidity of its action, in doses of from $\frac{1}{20}$ to $\frac{1}{6}$ grain three times a day. Bichloride of mercury has many adherents. The liquor of Van Swieten is the form recommended by Parrot for internal administration. The formula is as follows:

R \bar{y} . Bichloride of mercury	1 part.
Water	950 parts.
Rectified spirits	100 parts.

Sig. 5 to 20 drops in milk three times a day.

The bichloride of mercury may be given in simple watery solution, which may be combined with milk, and hence readily taken by the infant. The dose varies from $\frac{1}{200}$ to $\frac{1}{60}$ of a grain, according to the age and condition of the infant. If intestinal irritation be caused by the drug, a mixture of wine of pepsin and elixir of bismuth may be used as a menstruum.

An important element in the management of these cases will be the local treatment, applied to mucous patches, excoriations, and especially to the coryza. Ulcerations and destructive processes in the nose may be largely avoided by keeping the nasal passages clean by tepid water or bland oil. A 2 per cent. solution of the oleate of mercury will be efficacious in the nose. Mucous patches or condylomata should be kept clean, and may be dusted with calomel and bismuth. Nitrate of silver may be applied to patches appearing in the mouth that are intractable to internal treatment.

Where the bones are involved and evidence of gumma in any portion of the body is present, iodide of potash should be employed. In the visceral lesions this remedy likewise acts well; and if the indications arise, mixed treatment, by combining the biniodide of mercury with iodide of potassium, may be employed. The iodide of potassium is most efficacious, although the iodide of sodium may be administered with good results. The dose should be moderate, not averaging more than a few grains.

The general care of the nutrition of the syphilitic infant is most important. The chances for maintaining good nutrition are much improved by keeping the baby on its mother's breast. If the mother is unable to entirely supply the infant with nourishment, the bottle may be employed, but never to the complete exclusion of the breast. The well-known fact that an infant cannot infect the mother, although the latter shows no evidences of syphilis, justifies us in insisting upon her nursing her own infant. The employment of a healthy wet-nurse, although of advantage to the infant, is not justifiable, as the former will almost surely be infected by the latter. After nursing, the nipple should always be carefully cleansed, as well as the infant's mouth, by the use of some bland disinfectant solution. In cases in which the infant is deprived of the breast the most scrupulous care and cleanliness must be exercised in artificial feeding. A mild form of indigestion will severely handicap the syphilitic infant, and may eventuate in its death. General tonic treatment and stimulation may be employed in connection with specific treatment.

The treatment of the later forms of syphilis will depend upon the activity of the morbid process. Mercury should always be exhibited in some form when there is any evidence of active syphilitic disease. It has been proven that small and proper doses of mercury are tonic in syphilis, and actually relieve the hydræmia and defective nutrition so often seen in this disease. If there is no evidence of an active syphilitic process, the treatment will resolve itself into improving the nutrition of the child in every way. Good food, tonics, iron, cod-liver oil, change of air when possible, are all of value in aiding healthy growth and development in these retarded cases.

PART III.

THE INFECTIOUS DISEASES.

MEASLES.

By LOUIS STARR, M. D.,

PHILADELPHIA.

RUBEOLA is an acute, infectious disease, characterized by coryza and other catarrhal symptoms, by continued fever, and by an eruption of slightly elevated, crimson papules upon the face and body, followed by furfuraceous desquamation.

It is perhaps the commonest of the infectious diseases of childhood, and very few individuals arrive at adult age without having suffered from an attack. One attack usually protects against a second, though instances in which there have been two, or even three, attacks are not rare.

In large cities scattered cases of measles may be encountered at almost any time, but at certain recurring intervals, varying from eighteen months to two years, the disorder becomes epidemic. These epidemics are alike in the fact that young children, being unprotected by a previous attack, uniformly suffer most; unlike, in the extent of their prevalence, in fatality, and in the accentuation of particular symptoms. In isolated localities, having infrequent communication with large centres of population, and where measles has prevailed only at long intervals, the disease when it does arise finds a greater number of victims, attacks a larger proportion of adults, and is more fatal. When introduced to a virgin soil the virulence is extreme. As an instance of this the four months' epidemic of 1875 in the Fiji Islands may be cited: during it 40,000 natives died out of a population of 150,000—upward of 1 to every 4 souls. By contrast, the mortality in London in 1886—an average year—was 1 to each 2000 of the population.

Etiology.—The prime cause of the disease is a specific poison, the nature of which has not been determined, though A. Ransome and Braidworth and Vacher have discovered, in the breath and secretions of measles patients, certain peculiar organisms identical with those to be described as existing in the skin, the lungs, and the liver. It is certain, however, that the poison spreads by contagion, and most probable that, whether or no these micro-organisms carry it, it is given off in the breath and secretions. The contagion is usually conveyed directly from the sick to the well, and is so virulent that when once introduced to a dwelling or hospital ward its spread is rarely stopped until all unprotected inmates suffer. It may be carried from place to place by fomites, but simple airing of the clothing is usually sufficient to disinfect it. When such instances of infection occur close connection is shown, the medium being a child or nurse coming directly from an infected house. Experimentally, the disease has been propagated by inoculation with the blood, the nasal and bronchial mucus, and the tears of a patient, and also

with the serum taken from the vesicles which occasionally accompany the eruption. Infection begins in the incubative stage, is most active during the pre-eruptive period of coryza and fever, continues throughout the eruption, and thereafter rapidly subsides, to disappear at the end of the third week.

No age of infancy or childhood is exempt from measles. It may occur in sucklings a few weeks old, but is uncommon during the first six months of life. The period of greatest susceptibility is between the second and sixth years.

According to some authorities, males are more prone to be attacked than females, but the disproportion between the two sexes is insignificant. Season, too, seems to have little influence in furthering the onset of the disease. If there be any difference, it is in favor of the damp, changeable, depressing weather of March, April, and early May. In the Children's Hospital of Philadelphia, for example, scarcely a year passes in which there is not a more or less extended epidemic during these months. Apart from unknown atmospheric causes, the explanation may be found in the fact that at this season children are below par, or impaired in health by the disorders and confinement incident to the winter months, and therefore less able to resist the contagion which is always latent in large cities.

Pathology.—When death comes early in the course of the disease from the force of the poison itself, an autopsy reveals hypostatic congestion of the lungs, hyperæmia of the mucous membranes, and congestion of the organs generally, with extravasation into their substance, and softening. The blood is fluid, dark-colored, and deficient in fibrin.

During an epidemic at the Philadelphia Hospital, Drs. Keating and Formad detected large numbers of microbes in the liquor sanguinis and white corpuscles of blood taken from malignant cases, and the author has since made the same observation. Quite recently, too, a bacillus has been discovered in the urine of rubeolous patients. What relation these organisms bear to the disease cannot yet be definitely asserted. In sections of skin made on the sixth day of the eruption Braidworth and Vacher found swelling of the chorium and thickening of the rete Malpighii, due to great proliferation of cells which extended along the hair and sweat-ducts into the glands. Sparkling, colorless, spheroidal, and elongated bodies were also present in the true skin next to the rete, in the lungs, and in the liver. In each situation these bodies were mixed with others, spindle-shaped, staff-shaped, and canoe-shaped; all appeared to be albuminoid in character.

Other morbid appearances vary with the complications upon which death so frequently depends. The most common lesions are those of diffuse bronchopneumonia and of structural alterations of the mucous membrane of the gastro-intestinal tract, either catarrhal inflammation, follicular entero-colitis, ulcerative inflammation, especially of the colon, or softening. Less frequent are caseation of the bronchial glands, miliary tuberculosis of the lungs, pulmonary collapse, membranous laryngitis, diphtheria of the pharynx, and effusions into the pleuræ and other serous cavities.

Incubation.—The interval between the actual introduction of the poison and the appearance of the first symptoms of illness has been quite accurately determined—first, by experiment, measles having been introduced by inoculation in Edinburgh, Italy, and Germany; second, by the careful study of outbreaks in virgin soil, such as that in the Farøe Islands, by Panum; and third, by ordinary clinical observation. From all these sources the period may be fixed at from ten to twelve days.

Adults and older children may complain of distaste for food, slight head-

ache, and lassitude for several days before the actual beginning of the disease, but younger children appear to be perfectly well, and practically there are no symptoms during incubation.

Symptoms.—The course of rubeola may be divided into several stages.

Prodromal Stage.—This lasts about four days, and is characterized by the following group of symptoms: lassitude, irritability, at times chilliness, pain in the back and limbs, headache, loss of appetite, thirst and other indications of gastro-intestinal disturbance, and, more important, fever, with the various signs of catarrhal irritation of the mucous membrane of the eyes, nose, fauces, and larynx. The chilliness is not marked, rarely amounting to more than a disposition on the part of the patient to keep near a fire or a desire for more clothing, and a degree of coolness in the extremities appreciable to the nurse's hand. The same may be said of pain in the back and limbs, its presence in older children being established only by close questioning, and in younger by their showing indications of suffering when moved.

Pyrexia is uniformly present. It may be postponed until the second day of the prodromal stage, but usually begins on the first. The fever is continued in type, the *ascent* of temperature being marked by evening exacerbations

FIG. 1.

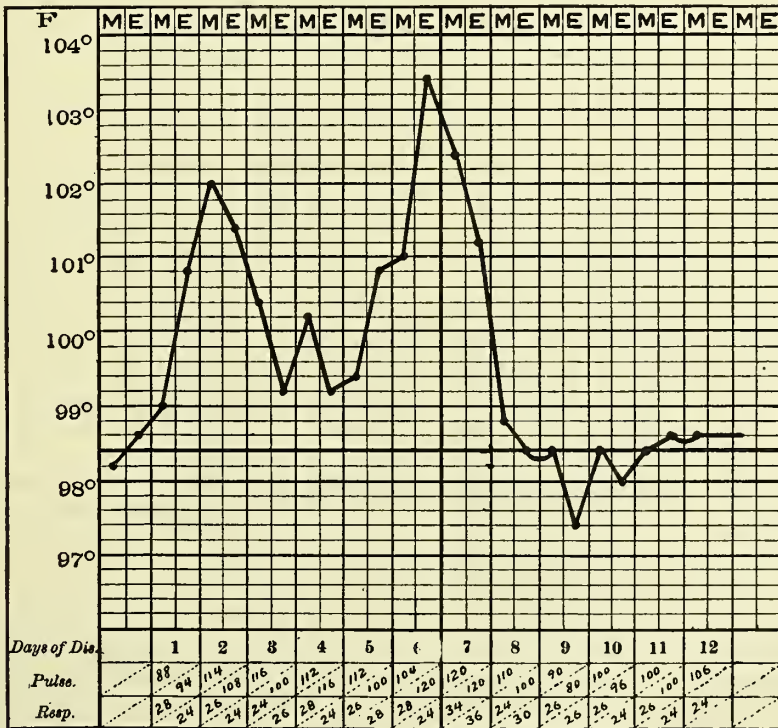


Chart of Temperature in Measles, showing Pre-eruptive Rise.

This chart was taken from a negro boy *æ*t. eight years, a patient at the Children's Hospital, Philadelphia. The attack of measles began on the day marked 1; the eruption was detected on that marked 5, and was at its height on 5 and 6.

(about 2°) and morning remissions (about 1°), which show a tendency to become less decided and shorter as the day of eruption is approached. Sometimes there is a marked remission or complete intermission on the second or third day, after which the temperature curve pursues the ordinary course. (See Figs. 1 and 2.)

With the rise in temperature the pulse becomes increased in frequency, force, and volume, though it is rarely as frequent as in scarlet fever. The skin, while moist, feels hot; complaints are made of frontal headache; and the child, at first irritable and restless, gradually passes into a condition of quiet and drowsiness, when it is said to "sleep for the measles."

The pathognomonic catarrhal symptoms begin with, or even precede, the pyrexia. These are inflammation and redness of the conjunctivæ—the palpebral portions especially—injection of the whites of the eyes, photophobia, lachrymation, stuffing of the nose, sneezing, and an abundant discharge of muco-purulent fluid from the anterior nares. The secretions from the eyes and nose are irritating and excoriate the skin over which they flow; the redness thus produced, with the injection of the eyeballs, the swelling of the lids and face generally, make up a heavy, almost characteristic, physiognomy.

Cough is usually present from the first day. Slight and infrequent in the beginning, it gradually increases, until on the third or fourth day it assumes a peculiar character. It is laryngeal, hard, dry, rather hoarse, and occurs in short paroxysms. Expectoration, when present, is slight and consists of clear, viscid mucus. The voice is hoarse.

The tongue is covered with a light white coating; the tonsils are moderately enlarged; the mucous membrane of the soft palate, fauces, and pharynx is uniformly swollen and reddened, and from twelve to twenty-four hours before the close of the prodromal period often becomes maculated with darker red, slightly-elevated spots closely resembling those of the cutaneous eruption; the latter appearance is most noticeable upon, and may be confined to, the soft palate.

Koplik recently called attention to a peculiar eruption upon the buccal and labial mucous membrane which he claims to be pathognomonic of measles. This eruption appears on the first day of invasion as a variable number of "small, irregular spots of a bright-red color," each having in its centre a "bluish-white speck." As the skin-rash appears the eruption on the mucous membrane grows diffuse, and when the former is at the efflorescence the latter has but the characters of a discrete spotting, and has become a diffuse redness with innumerable bluish-white maculæ scattered over its surface. This symptom must not be confounded with the pharyngeal eruption already mentioned, and, if as constantly present as Koplik asserts, will prove of great diagnostic value in the early stage of the disease.

Moderate enlargement of the glands behind the angle of the jaw is an ordinary feature, and the same condition of the cervical lymphatics may sometimes be observed.

There are anorexia, thirst, slight difficulty in deglutition, sometimes vomiting, and at first constipation, later diarrhœa.

Of nervous manifestations, irritability and drowsiness have been already mentioned. The latter symptom is often very marked, the child sleeping for the greater part of one or even two days before the rash appears, waking only to ask for drink or to have its urgent wants attended to, and then drowsing off again. There is no danger in this condition, unless it be associated with indications of cerebral disease or deepen into coma or alternate with decided delirium. Restlessness with mild delirium at night may take the place of drowsiness, and, in exceptional cases, convulsions occur.

Eruptive Stage.—The eruption usually appears in the evening of the fourth day. For a few hours immediately preceding its outbreak the nervous symptoms are increased, or, if absent before, are developed, and it is at this time that convulsions are most liable to take place. The rash shows itself first

on the skin immediately behind, beneath, and in front of the ears; thence it spreads to the rest of the face, the neck, the trunk, and the limbs, completing its extension over the entire body in from twenty-four to forty-eight hours. It begins in the form of distinct maculæ, more or less deep crimson in color, rounded in shape, with irregular edges, and varying from half a line to three lines in diameter. These soon develop into slightly elevated papules with hard, flat summits, which feel firm to the touch and temporarily lose their color under pressure. Isolated and few in number in the beginning, the papules rapidly become more abundant, and show a tendency to arrange themselves into irregular clusters, the unaffected portions of the skin preserving the normal appearance. The intensity of the eruption varies greatly; sometimes the papules are

FIG. 2.

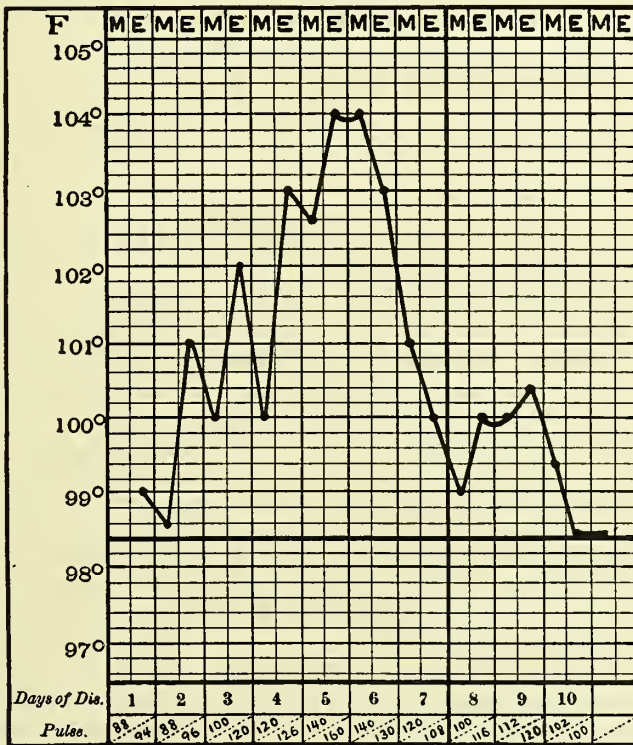


Chart of Temperature and Pulse in Measles.

quite scattered and the few clusters are separated by large areas of healthy skin; at others they are so numerous and coalesce so closely that extended portions of the surface assume a dark-red tint. This coalescing is most frequently observed on the face, on the neck and back, and near the flexures of the joints. Occasionally, in very severe cases, minute vesicles form on the summits of the papules. After full development the rash shows little change for one or two days. It then begins to fade in the order of its appearance, assuming a lighter or yellowish-red color, and in a day or two later disappears, leaving only faint reddish stains which mottle the skin for several days longer. The subsidence of the rash is followed by desquamation, the epithelium falling in very fine bran-like scales. This process is most noticeable on the face, but even in this position may readily escape observation.

The rash may vary in other characters as well as in its intensity. Some-

times the papules on their first appearance are hard and prominent, resembling closely those of variola. Again, their crimson color may not entirely disappear on pressure—a condition due to great hyperæmia of the skin. Finally, the eruption may steadily grow darker until a deep-purple color is acquired; this is also due to intense hyperæmia with rupture of distended cutaneous capillaries. Such a rash does not disappear on pressure: it remains at its height much longer than the ordinary eruption, and is slow in fading.

The fever does not abate on the appearance of the eruption; on the contrary, it often attains a higher marking (103° – 105° F. in the axilla) on the first and second day; after that, as the rash fades, it rapidly falls to the normal line.

The preceding chart (Fig. 2) presents a fair picture of the temperature curve of measles of average severity. The patient who furnished the record was a boy five years old, an inmate of the Children's Hospital, Philadelphia. Having been directly exposed to contagion, the symptoms of coryza were noticed on the day marked 1: the eruption appeared on the evening of that marked 4, and was at its height on 5 and 6. Afterward the eruption rapidly faded, and with it the temperature fell almost to the normal line on 8, though complete lysis was delayed for forty-eight hours by a trifling secondary laryngeal catarrh.

The pulse increases in frequency as the temperature rises, and follows its curve moderately closely. The maximum ratio is usually about 120 beats per minute, though it occasionally rises higher, as in the case just referred to.

During the acme of the eruption and pyrexia the catarrhal symptoms become more severe. The conjunctivæ are red, the eyelids are much swollen, photophobia is extreme, and there is a copious flow of irritating tears; the nasal passages are dry and encrusted, or there is a free discharge of acrid mucus, and crusts of dried blood may often be seen about the nostrils, for epistaxis is common. The upper lip is tumid and excoriated, the cheeks are swollen and deeply reddened, and the characteristic physiognomy, already mentioned as existing in the prodromal stage, is more strikingly marked. The tongue is usually moist, with a thick, yellowish-white central coating and red tip and edges; the soft palate, tonsils, and pharynx are red; and the throat feels sore. Thirst and anorexia continue; there may be some tumefaction and tenderness of the abdomen; moderate diarrhœa is the rule; and in some cases there are violent vomiting and purging. The respiratory movements are somewhat quickened: the voice is husky, the cough is paroxysmal, dry, hoarse, and troublesome, and attacks of spasmodic croup are apt to occur. Physical examination of the chest reveals the signs of catarrh of the larger bronchial tubes, and as a rule—especially in scrofulous children—of enlargement of the bronchial glands. The probability of a similar enlargement of the glands at the angles of the jaw and sides of the neck must also be remembered. The urine is scanty, dark yellow in color, with abundant urates, and, while the temperature remains elevated, may contain a trace of albumin. Prostration of the general strength is not decided in the majority of cases.

Stage of Decline.—So soon as the rash begins to fade—fourth day of eruption, eighth of disease—the other symptoms rapidly abate. The pulse loses its rapidity, though it is somewhat weaker than normal; the temperature steadily falls, often with considerable sweating; the coryzal symptoms subside; the voice becomes less hoarse; the cough grows looser and less frequent; and, if the child be old enough, nummular masses of muco-purulent matter are freely expectorated. The tongue cleans off; appetite returns; there is no

longer thirst, irritability, or restlessness; the bowels return to their normal condition, and ordinary health is soon regained.

Modified Forms.—Measles without eruption and measles without catarrh have been described by different authorities. In regard to the first modification, it is difficult to doubt the records of certain isolated cases that have occurred during epidemics of the disease, though the author has never met with any examples. On the other hand, cases reported as "*rubeola sine catarrho*," must be classed under rubella rather than modified rubeola.

There is, however, a form of measles which is distinguished from its outset by typhoid symptoms, and is very fatal. Malignant, ataxic, or black measles, as this variety is called, may occur as an epidemic or sporadic affection, but it is usually the former. There is great prostration; the patient is dull and stupid; the pulse is small, feeble, and frequent; the respiratory movements are difficult and rapid; the rectal temperature is high, often reaching 107° or 108° F., while the hands and feet feel cold; the tongue is dry, brown, and thickly coated; epistaxis is often obstinate, and hæmaturia may occur. The rash appears slowly, imperfectly, and irregularly, assumes a livid, purplish, or blackish hue, and may quickly retrocede; at the same time, the skin is thickly mottled with petechiæ. The attack progressing, the pulse becomes so rapid that it can scarcely be counted; there is muscular tremor with muttering delirium, and life terminates in coma or convulsions. After death ecchymoses may be found in the viscera.

Complications.—The conditions which disturb the regular course and threaten the ordinarily favorable result of measles are mainly furnished by an undue development of certain of the usual or unusual features—an exaggeration determined either by the nature of the special epidemic or by certain constitutional peculiarities of the individual affected. These complications may be described in the order of their frequency and importance.

Bronchial catarrh may spread from its ordinary position, the larger tubes, to those of smaller calibre, and become a grave complication. The extension is most common in infants under one year, and in them usually proves fatal through collapse of the lung—a condition readily produced at this early age. The indicative symptoms are dyspnoea and rapid breathing, lividity of the face and extremities, a haggard and anxious expression of the countenance, and the detection, on auscultation, of fine subcrepitant râles distributed throughout both lungs.

After the age of twelve months catarrhal pneumonia is more frequent than extended bronchitis. It is, in fact, the most common complication of the disease, and may occur at any time during its course. When it arises early, the eruption is often delayed, or, if already present, may retrocede, and there is considerable aggravation of the general symptoms. If later—at about the time of the disappearance of the rash, for example—the temperature, instead of falling, remains high, ranging in the neighborhood of 102° F.: in place of the usual general improvement, there are greater weakness and more manifest illness; the patient is listless and takes little interest in his toys or in what is going on about him; there is increased thirst and anorexia; the face is pinched and distressed-looking; the lips are livid, and the alæ nasi move to and fro with the breathing, which is labored and quickened. On physical examination of the chest the ordinary signs of broncho-pneumonia can be detected. This complication varies greatly in degree of severity. It often runs a prolonged, subacute course, and may terminate in complete recovery, in death, or, becoming chronic, may merge into one of the varieties of pulmonary phthisis.

Intestinal catarrh, which is usually productive of nothing more than a

trifling, readily-controlled diarrhœa, may be aggravated into an entero-colitis, or even an ulcerative inflammation of the mucous membrane of the colon. These complications are excited by improper food, by injudicious use of purgative medicines, and by careless exposure to cold and dampness. They sometimes appear during the initial stage, but are usually developed later in the disease. The symptoms are tumidity and tenderness of the abdomen, colic, tenesmus, and more or less frequent purgation, the evacuations being green in color and containing glairy or bloody mucus. The regular course of the disease is little affected, though in nervous, sensitive children the intestinal lesions may maintain a temperature of 104° or 105° F. for several days after the subsidence of the rash. In such cases convalescence is prolonged, though the ultimate outlook is favorable unless catarrhal pneumonia coexists; then the danger inherent to the latter condition is greatly increased.

Laryngitis often complicates measles. It is most likely to occur during the decline of the eruption. Ordinarily the spasmodic form—false croup—is assumed, with symptoms that are alarming to the uninitiated, but really devoid of actual danger and without effect upon the regular course of the disease. Sometimes, on the contrary, a pseudo-membranous exudation forms in the larynx, and the case at once becomes extremely grave. The symptoms are the same as in idiopathic cases. Thickening, softening, and ulceration of the mucous membrane occasionally occur, and Rilliet and Barthez record a case in which suppuration about the larynx followed an attack of measles.

Convulsions happening during the eruptive stage are of grave import; preceding it, they are seldom serious.

Epistaxis, when it becomes profuse and exhausting, always tends to postpone the restoration to health, and may determine death in weak subjects or when the disorder is severe and ataxic in type.

Ophthalmia and otitis are infrequent complications, and are almost entirely limited to patients having tuberculous tendencies. Both yield sluggishly to treatment, and otitis may prove fatal by an extension of the inflammatory process to the membranes of the brain.

Paralysis should be mentioned as a rare accident that may be associated with measles. Drs. Barlow and Ormerod have recorded cases in point.

Sequelæ.—Many of the conditions referred to as complications may also occur as sequels of the disease. Thus catarrhal pneumonia, laryngitis, and bronchitis in chronic form, and chronic gastro-intestinal catarrh are frequent results. Enlargement of the bronchial glands is another common sequence, and acute tuberculosis so often follows that the physician must suspect its development whenever a patient remains feeble and feverish after an attack of measles. In children having a tuberculous diathesis the disease is very prone to light up any or all of the troubles which are characteristic of their constitutional taint. Other less common sequelæ are “marasmus,” or a condition of general wasting and debility; diseases of the eyes and ears; ulcerative stomatitis, with necrosis of the jaw; gangrene of the cheek and vulva; necrosis of the nasal cartilages; and, rarest of all, renal disease.

Whooping-cough is generally supposed to bear an intimate relation to measles. Epidemics of the two diseases undoubtedly often follow close upon each other without any uniformity of precedence. What the actual connection may be is uncertain, but it is probable that the presence of one exanthem merely lessens the resistance which a healthy body manifests to the infective power of the other.

Diagnosis.—The distinguishing features of rubeola are the long prodromal stage with its marked catarrhal symptoms; the course of the fever-curve, espe-

cially the continuance of high temperature for two days after the appearance of the eruption; and the peculiarities of the rash. It should be remembered, however, that the rash, though quite characteristic in typical cases, is more apt to be misleading, through its variations, than any of the other pathognomonic signs; and it may be said of measles, as indeed of all other exanthemata, that a diagnosis must never be based exclusively upon the eruption.

In the initial stage it is often difficult to differentiate between measles and an ordinary acute catarrh—a “severe cold.” The coryzal symptoms are identical: hoarseness and cough are present in both, and both are attended by fever. If such symptoms are developed at a time when measles is epidemic, the probabilities are strongly in favor of an attack of the disease. On the other hand, if the history of exposure to contagion is uncertain, it is best to withhold a decided opinion and wait for the appearance of the rash, which, it is well to recollect, shows upon the soft palate from twenty-four to forty-eight hours before it can be detected upon the skin. In this connection the buccal eruption described by Koplik, and already mentioned, may be of great assistance in establishing an opinion. It may be stated here that this element of uncertainty in the early diagnosis is much to blame for the ready and wide extension of the disease; for, while contagion is freely given off by patients in the catarrhal stage, isolation is rarely practised until all doubt as to the nature of the attack is cleared up by the eruption.

Sore throat, which is sometimes present, combined with fever, may suggest scarlatina, but the latter disease has a sudden onset, with vomiting, rapid and extreme elevation of temperature, and very frequent pulse, and without catarrhal symptoms; further, the characteristic eruption appears not later than twenty-four hours from the commencement of the attack.

In the eruptive stage, when the color and grouping of the papules are typical, and the fever, coryza and cough marked, there is little room for error.

When the rash appears in hard, isolated papules, variola may be suspected, a mistake not uncommonly made. In small-pox, however, the pre-eruptive stage is characterized by obstinate vomiting and severe pain in the back. When the eruption appears, the temperature abruptly falls and the active symptoms abate; the papules themselves are harder than ever noticed in measles, feeling like pellets of shot under the skin, and by the second day those first appearing on the face are changed into vesicles.

There is more difficulty in distinguishing the rubeolous eruption from the rash of rubella than from that of any other of the exanthemata. The points of distinction are the short, often featureless, prodromal stage of rubella, the comparative absence of catarrhal symptoms, and the fact that the papules are smaller and lighter in color, appear almost simultaneously on the face, the wrists, and the ankles, and thence extend over the body, showing no tendency to irregular grouping.

Various skin eruptions, notably the early stages of acute and general eczema and syphilitic roseola, resemble the rash of measles, but the differences in clinical history and the entire absence of general symptoms render the distinction easy.

Prognosis.—Generally speaking, the percentage of fatality in rubeola is small. Nevertheless, in individual cases the prognosis depends upon the type of the epidemic, the age and previous condition of health of the patient, the nature of the hygienic surroundings, and the character and severity of the complications.

An attack, of whatever severity short of malignancy, occurring in a previously healthy child over the age of two years, who is surrounded by the usual

comforts of life and treated with ordinary skill, should almost invariably terminate in recovery; and in such cases even the onset of so serious a complication as catarrhal pneumonia is rarely fatal. Quite the reverse is true when the disease attacks children who are constitutionally feeble or debilitated by some antecedent acute disease, who are suffering from rickets or suppurative bone disease, who have chronic pulmonary lesions, who are subjects of the tuberculous diathesis, and who live in crowded and filthy houses or unhealthy localities. These patients, when they survive the force of the disease itself, are often carried away by one of the complications or sequelæ, to the development of which they are very prone.

In children under two years of age measles is more serious, and the younger the infant the greater is the danger of an unfavorable termination. Here death is due to the readiness with which bronchial catarrh extends to the finer tubes, producing catarrhal pneumonia or pulmonary collapse—a tendency inherent to every catarrh in the very young, but most marked in that attending measles, and very apt to be exhibited in weakly or rachitic infants.

The gravity of the different complications and the fatality of epidemics of malignant type have already been referred to. In ordinary epidemics the prognosis becomes unfavorable under the following conditions: When the prodromal stage is more prolonged than usual and attended by violent symptoms of any kind, as great jactitation, irritability, dyspnoea, stupor, and coma or convulsions; when the eruption is irregular in development or course; when the pyrexia continues after the subsidence of the rash; when in the later stages of the disease the face remains deeply flushed or grows pale; when cough, dyspnoea, or diarrhoea persist, and when the child is left weak, languid, dispirited, or irritable.

Dr. Ellis places the mortality of measles at 1 in 15 cases. My own experience has been much more fortunate. In private practice all of my cases have recovered save one, and that, an infant of nine months, died of meningitis directly due to the active lighting up, by the measles, of a long-standing disease of the middle ear. Even in my hospital wards the mortality has been less than that given by the author quoted, and the deaths, while occasionally due to the force of the poison on enfeebled bodies, have mainly occurred in patients previously affected with spinal caries or suppurating joints or having badly deformed rachitic chests.

Before leaving this division of the subject some attention should be given to the question of the liability of the *return* of measles. The fact is, that, next to typhoid fever, measles is the most liable of all the exanthemata to return. A number of cases are on record in which patients have had a second attack after a short interval, and sometimes so soon after the first as to constitute a true relapse, both attacks running their course within a period of four or five weeks.

Treatment.—Attention must be directed first to the hygienic management of the disease, as this is of vast importance in all cases, and in those of ordinary severity suffices, with a very little aid from simple drugs, to ensure a favorable ending.

As early as the nature of the attack can be decided upon the patient must be put to bed, and confined there until not only the rash itself, but all traces of the remaining yellowish-red stains, have disappeared—about the eighth or tenth day of the disease. Young infants, with whom it is difficult to enforce complete rest in bed, must, when taken up, be held upon the nurse's lap and be properly protected by some light wrap. If it be possible to have two cots, one for day and the other for night use, the patient's comfort is greatly

increased. Care must be taken to provide only sufficient bed-covering to maintain warmth; the mattress should be of hair, and, when only one bed is at command, the sheets ought to be changed at least once each day, though accidental soiling may render more frequent renewal necessary. A large, airy, and, if possible, isolated chamber is to be selected for the sick-room, and an open fireplace for wood or coal is the best method of heating, at the same time securing free ventilation without draughts. When heat is supplied from a furnace, change of air must be effected by a window or door, the patient being protected from chilling currents by a carefully placed screen. The proper temperature is 65° to 68° F. During the continuance of photophobia and conjunctival irritation the room must be moderately darkened, and it is always well to see that the bed is so placed that the patient's face will not be turned directly toward a window. All superfluous hangings and furniture should be dispensed with, though it is unnecessary to strip the apartment so completely as in case of scarlet fever.

After the child is well enough to leave his bed he should be kept in the sick-room for three or four days; then, so far as his own safety is concerned, he may be allowed the range of the house, but not permitted to go out doors for a week longer, and then only in favorable weather. If, however, there are other susceptible subjects in the house, and the question is one of isolation, he must not quit his chamber until the end of the third week from the beginning of the attack.

The diet requires careful regulation. Nursing infants must be fed, during the febrile stage of the disease, at somewhat shorter intervals than in health, but if, on account of increased thirst, they suck very greedily, the time of lying at the breast must be curtailed, the object being to secure sufficient nourishment without at any time overloading the alimentary canal and overworking the digestive powers, which are enfeebled by the catarrhal condition of the mucous membrane. With bottle-fed babies it is even more essential to carefully regulate the administration and preparation of the artificial food. For example, a child of nine months, who in health would be fed five times daily and take in all about forty fluidounces of appropriately strong food, must during measles be placed nearly on the plane of a child six months old, the feedings being increased to six or eight a day, the total quantity reduced to thirty or thirty-four fluidounces, and the strength proportionally lessened. For the purpose of dilution lime-water or barley-water may be employed with advantage, on account of its power of preventing rapid coagulation and the formation of large, tough curds in the stomach.

Should ordinary milk mixtures disagree, it is well to resort to Pasteurization or partial predigestion, and if it be impossible for the infant to retain any form of milk food, as is sometimes the case, raw beef juice in doses of two teaspoonfuls every two hours, or veal broth and barley-water may be resorted to as temporary substitutes.

Patients who are old enough to take a mixed diet when well should at once be placed upon liquid food.

To relieve thirst, pure water, carbonic-acid water, and Vichy are preferable to any of the old-fashioned sweetened or acidulated drinks. They are to be given cool (not iced), and in moderate quantities at short intervals. In administering drink a good plan is to use a small glass—holding a fluidounce, for example; to drain this gives the child more satisfaction than the same draught from a larger vessel which he is not allowed to empty, and there is much less danger of an excessive quantity being taken.

With the decline of the temperature and the abatement of symptoms denot-

ing gastro-intestinal disturbance, additions may gradually be made to the diet until the full feeding of health is resumed.

Due attention must be paid to keeping the patient's person clean. To this end the face, hands, portions of the body liable to become soiled, and even the whole surface, should be sponged with tepid water every morning, each part being washed and dried separately, so as to avoid exposure and chilling.

When the patient is well enough to go into the open air, it is essential to see that he is properly dressed with warm woollen under-clothing; morning spongings with salt water may also be ordered now, and complete restoration to health will be greatly hastened by a change of air. So the atmosphere be dry and bracing, it makes little difference, in ordinary cases, whether the resort selected be at the sea-coast or inland, though the former is to be preferred when the disease leaves the subject with marked glandular enlargements or develops other manifestations of the tuberculous diathesis.

The medicinal treatment of ordinary cases of measles is very simple. Early in the attack, while the temperature is elevated and the cough hoarse, citrate of potassium is useful as a febrifuge and relaxing expectorant. To a child six years old from one to two fluidrachms of liquor potassii citratis should be given every two hours, and to this may be added 20 drops of paregoric and 5 or 10 drops of syrup of ipecacuanha if the cough becomes very troublesome and croupy—a tendency often exhibited during the first two or three nights of the attack. Later, as the cough grows loose, a stimulating expectorant should be substituted. The best of this class of drugs is chloride of ammonium, which must be given in solution and in doses of 1 to 2 grains every second hour. As convalescence approaches the expectorant may be gradually discontinued, and 1 grain of quinine may be given three times daily, either in solution or in chocolate tablets; sometimes, too, there is sufficient debility to warrant the administration of moderate doses of whiskey. Finally, a course of iron or of cod-liver oil—in tuberculous cases—is often necessary.

While pursuing these general measures the eyes need careful attention. Four times daily the lids should be washed with water as hot as can be borne, and afterward a few drops of a solution of borax (gr. v to f̄3j) gently applied to the conjunctivæ. In case of great photophobia and conjunctival irritation a weak solution of cocaine (gr. j to f̄3ss) may be dropped into the eye twice daily. It is well also to spray the nares and pharynx at frequent intervals with Dobell's solution or Listerine diluted with water (1 part to 6), or, if the patient be old enough, the throat may be gargled every three hours with one teaspoonful of chlorate of potassium dissolved in 4 fluidounces each of claret and water. Mild counter-irritation of the skin of the throat is often of service in relieving pain and hoarseness; for this purpose a combination of turpentine and olive oil (1 part to 2 or 3) may be employed several times in the twenty-four hours.

Malignant measles demands a stimulant and tonic treatment. Whiskey or brandy in properly proportioned quantities must be added to the milk, or brandy-and-egg mixture may be employed, and raw beef juice and concentrated meat broth must form an element in the diet. Of drugs, quinine, carbonate of ammonium, and digitalis are called for, and must be used in sufficient doses to meet the urgency of the indications. In this form mustard baths and hot packs are of great service. For the mustard bath, which is more suitable for children under three years of age, the water should be at a temperature of 100°, and contain about one tablespoonful of mustard to the gallon; the patient is immersed up to the neck for three minutes, then quickly

dried and placed in bed between blankets or wrapped in a blanket and dried later. The bath may be repeated in two hours if necessary. In hot packing the child is placed between blankets, and then a blanket wrung out as dry as possible, after being wet with hot water or mustard and water (two teaspoonfuls to the gallon), is quickly wrapped about the body, care being taken lest it be too hot; it may be renewed in half an hour.

At times one or more of the symptoms of the disease may be so modified or exaggerated as to require special treatment.

Headache, when violent, is usually attended by constipation, and can be relieved by unloading the bowels and by putting the feet in hot mustard-water (one tablespoonful to the bath) or applying a mustard plaster (1 part to 4 or 6 of flour) to the nape of the neck. For the purpose of evacuating the bowels enemata or glycerin suppositories should first be tried, and if these fail, a mild laxative, as calomel in broken doses or milk of magnesia with aromatic syrup of rhubarb, may be administered. Active purgatives should never be employed, on account of the decided diarrhoeal tendency of the disease. Should these measures fail to relieve the headache, resort must be had to bromide of potassium or elixir of the valerianate of ammonium.

Moderate looseness of the bowels need not be interfered with, but if the purging be sufficiently violent and continuous to threaten the strength of the patient, a combination of rhubarb, bismuth, and chalk mixture may be prescribed, or, if the evacuations be very watery, it may be necessary to use a more powerful astringent, as oxide of zinc in doses of gr. $\frac{1}{4}$ — $\frac{1}{2}$ every three or four hours.

Distressing vomiting is best treated by causing the patient to drink tepid water, and, when the stomach has been relieved of altered food and irritating secretions, applying weak mustard plasters to the epigastrium. In this condition, however, it is most important to pay careful attention to the feeding.

When the eruption is delayed, appears irregularly, or retrocedes, it must be remembered that the condition depends upon some complication—bronchopneumonia, for example—and that the true mode of relief is to relieve the internal inflammation which is the cause of the difficulty: hot mustard foot-baths or full baths, hot packs, mustard sinapisms, and stimulants are required. Liquor ammonii acetatis is a useful preparation in these cases; it may be given in doses of one to two teaspoonfuls every two hours. When the rash itches or burns, frequent applications of fresh lard or vaseline will afford relief.

At the acme of the eruption the temperature often runs up to 104° or 105° F. for a few hours, without corresponding severity of the other symptoms. No interference is necessary for a temporary elevation of this sort, but for a persistently high temperature of twelve hours or more some antipyretic must be given or cooling baths resorted to. Antipyretics are still on trial, but the safest is phenacetin. This may be administered in an initial dose of 1 grain for any age between two and six years. If the temperature falls afterward, wait and observe the extent of the depression; if not, repeat the dose after the lapse of an hour; should this fail, gradually increase the amount to 2 or 3 grains. The first dose may be given when the temperature ranges above 103°, and the drug may be repeated as often as necessary to keep it below this point, the cardiac condition being carefully watched in the mean time.

When baths are employed to reduce the pyrexia, water at a temperature of 95° to 98° F. should first be used; if this fail, tepid or cold spongings may next be resorted to, and as a final resort the tepid or cooled bath may

be tried. In giving the latter the child should be undressed as quickly as possible, and then immersed in a bath of 90° F.; cold water is now rapidly added until the temperature of the bath is reduced to 80°. After a sufficient intermission—usually five or six minutes—the body is quickly dried with a soft towel and the patient put back to bed between sheets. The effect of the bath is sometimes very powerful, and the child remains livid-looking and collapsed for some time. In such case small doses of brandy must be given in warm milk at short intervals and artificial heat applied to the feet.

It is stated by some authorities that antipyretics ought to be employed whenever the temperature reaches 102° F. Such a rule is dangerous. There are many instances in which, with a temperature of 102°, the child is very ill, and this degree of fever may be judged to be more than usually detrimental. For these a bath, either tepid or cold, cold sponging, or phenacetin, may be recommended, but for one such case there are many others that run a perfectly favorable course with a temperature even higher than this, and in which it is difficult to see what benefit could have accrued from antipyretics. Each case must be treated upon its own merits.

When in doubt as to the propriety of using antipyretic drugs or baths, it is well to try the effect of moderately full doses of sulphate of quinine. It has been my own experience that this agent given by the mouth, or, better still, by the rectum, in suppositories of two to four grains every three or four hours, frequently reduces temperature, and, should there be much associated restlessness, produces sleep.

The treatment of convulsions, broncho-pneumonia, and other disorders which may be associated with or follow after measles does not differ from that employed when these affections occur idiopathically, and therefore requires no especial consideration here.

Quarantine.—The rubeolous patient should keep his bed for eight or ten days and his room for three weeks; then, if he be quite well in every respect, there is little danger in his mixing with his playmates. When one member of a household is attacked, it is necessary for the other children of the family who have not had the disease to stop going to school or associating with other children, as it is probable that they also have contracted the malady, and, as it is infectious in its early stages, they may readily be the means of giving it to others. For the same reason it is unwise to send them away from home; at the same time they must not come in contact with the case already developed.

The convalescent should have a warm bath and fresh clothing before mingling with his associates. Scalding of the bed- and body-clothing and thorough airing and cleaning of the sick-room are all that is necessary in ordinary cases, though in malignant epidemics disinfection of the bedding and thorough fumigation of the chamber with sulphur should be insisted upon.

SCARLET FEVER.

By MARCUS P. HATFIELD, M. D.,
CHICAGO.

SCARLET FEVER, or scarlatina, is a self-limited, contagious, microbic disease, characterized by fever, angina, and a typical eruption, and followed by desquamation and recovery in about three weeks if the disease be uncomplicated.

The health reports of all of our large cities show that scarlet fever is an endemic disease of childhood, never being entirely stamped out, and affecting now only a trivial percentage of the population, and then increasing into epidemics of frightful mortality, often from causes as yet unknown to modern science.

According to Busey, it is the most widely disseminated of the exanthemata of childhood, and, perhaps rightly, the most dreaded of all the diseases of children, whose susceptibility varies not a little with their age. Infants under six months, as a rule, escape; 64 per cent. of all cases occur in children under six years of age (Murchison), after which susceptibility diminishes, though liable to as yet inexplicable variations, for children and nurses who have escaped half a dozen epidemics may succumb to the seventh after exposure apparently in no wise different from that which preceded it.

One attack, as a rule, protects from a second, though well-attested returns are on record. The majority of those cases popularly reported as second attacks are usually due to errors in diagnosis. But it must also be remembered that frequent abortive attacks of sore throat are well known to occur in nurses or physicians attending cases of this disease.

Scarlet fever may be complicated with other of the exanthemata, especially varicella. Cases of coincident scarlatina, variola, and measles are reported by Vogel.

While the disease is not so infectious as measles, as shown by the fact that 42 per cent. of Budert's unprotected children escaped infection during an epidemic in the isolated German village of Neundorf, it should be remembered that the contagiousness of scarlet fever varies greatly with the epidemic.

Brush's statement that the colored race possesses an immunity from this disease is erroneous, for the writer has seen scarlet—or rather royal purple—fever in a coal-black pickaninny, and in Chicago, at least, colored children enjoy like privileges in this respect with those of lighter skin.

History.—It is more than probable that scarlet fever must have existed as far back as there have been masses of people crowded together in great cities; but there are no earlier accounts of the disease than those of the seventeenth century (1610–18), when epidemics occurring in Spain and Italy were described by Mercatus, Heredia, and Symbatus (Bohn). About the year 1625 both sporadic and epidemic cases were met with in Breslau and described by a Dr. Doring, who is probably entitled to the honor of being the first German author to write on this subject. He was closely followed by Sennert's description of

the disease at Wittenberg, later followed by like outbreaks at Brieg (1642), Schweinfurt (1652), and in Poland (1664).

Up to the time of Sydenham scarlet fever was supposed to be a variety of measles, being known by such fanciful terms as "ingrassius, rosalia, rubeolas, morbilli ignei," etc. During the years 1670-75, Sydenham had ample opportunity to study the epidemics raging in the city of London, and differentiated the disease from measles. The origin of the name is yet uncertain (Bohn).

To Fothergill (1750) justly belongs the credit of establishing the contagiousness of scarlatina, and the facts upon which depend all modern theories of its prophylaxis. But many writers believe that the disease has steadily increased in virulence, until to-day it is the most prevalent and dangerous of all the diseases of childhood.

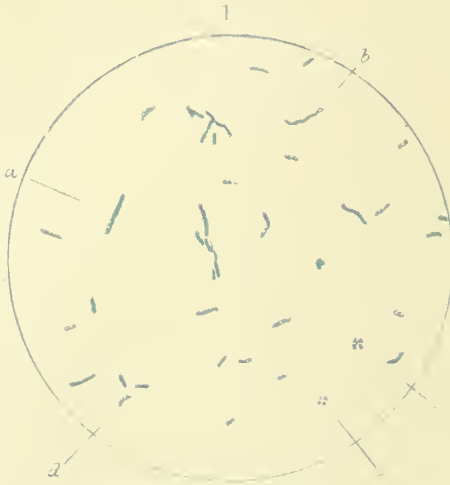
Scarlet fever is supposed to have been brought to North America in 1735, spreading slowly from the coast inland, and so infrequently met with that Dr. Rush, as late as the beginning of the present century, wrote: "No physician would be likely to see it more than once in his lifetime." At first it was regarded as rather a trivial affection, but malignant epidemics swept through Kentucky and Ohio when the country was almost an unbroken forest. Then came a period of slight malignity, so that Professor Chapman of the University of Pennsylvania so late as 1833-36 positively denied the contagiousness of this disease.

Etiology.—He would be a purblind physician who, in these latter days, would attempt to deny the microbic origin of scarlet fever, but it must as frankly be admitted that our knowledge concerning its exact etiology is as yet indefinite and conflicting. Klebs figures the peccant microbe and names it *Monas scarlatinosa*. Ecklund of Stockholm minutely describes another, which he is certain is the cause of scarlet fever, and proposes the name *Plox scindens*, a fuller description of which may be found under the heading of Pathology. Edington of Edinburgh later isolated from the blood and epidermic scales of scarlet-fever patients another microbe, which he and Dr. Shakespeare of Philadelphia unite in declaring to be the specific cause.

But, while it is disheartening that as yet we know so little accurately concerning the bacteriology of scarlatina, there is much that is well known and proven beyond dispute in regard to the spread of the disease and the nature of its contagion. First of all, it can be insisted upon that its *contagium vivum* is easily portable, tenacious in its power to do evil for years, and with great probability originating in some of the lower animals. The horse, the dog, and the cow all have had their claims advanced as first owners of the scarlatinal microbe, and during the Hendon epidemic some years since it seemed as if the question had been decided in favor of the cow. Later and more accurate investigations, however, seemed to show that the disease carried from the diseased teats of the infected cows was scarlatinal only in the form of the rash communicated to human beings.

There is also considerable dispute as to which of the secretions may carry the scarlatinal virus. Some writers insist that the patient is a source of infection from the initial sore throat until the last branny scales have dropped away from between the fingers and toes; others, that infection may be carried so long as there is a specific otorrhœa. Undoubtedly, the micro-organism usually enters the system by inhalation, but there seems to be good reason for believing that it may be taken in with food (Smith), or carried from person to person by inoculation of scarlatinal blood or blood-serum. It is, however, generally conceded that a scarlet-fever patient is most dangerous during the stage of desquamation, and that the branny scales of this period

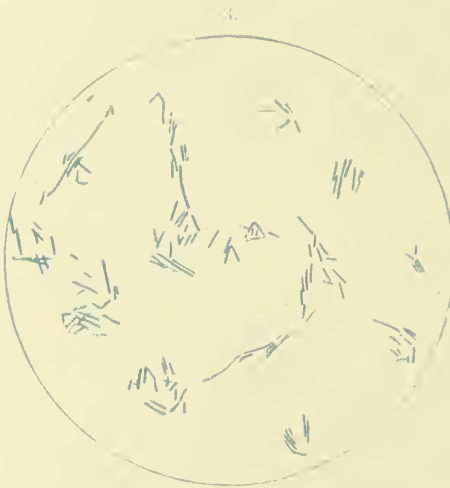
PLATE V



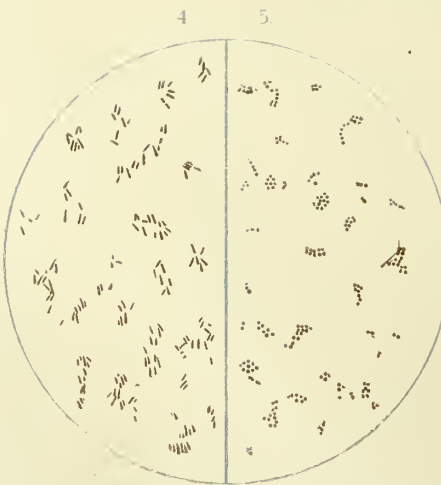
Original impure cultures from skin
1000



Original impure cultures from skin
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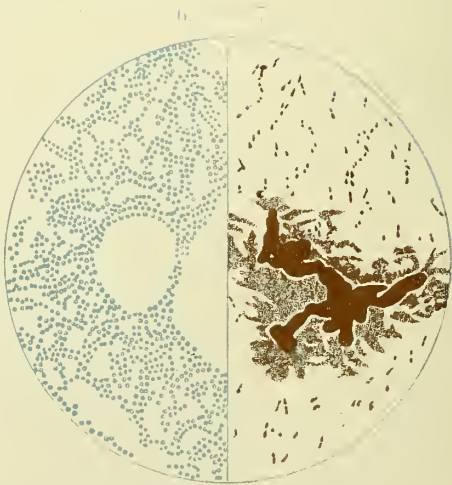
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Bacillus Fulyus

Streptococcus Rubiginosus

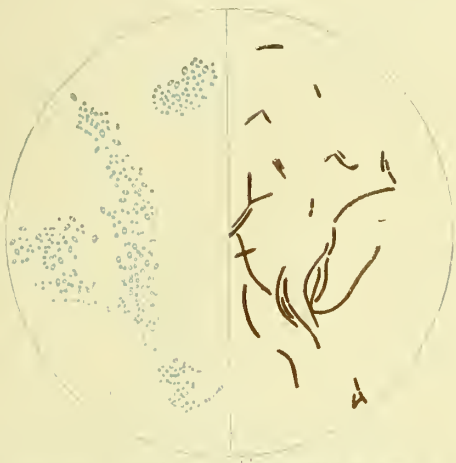
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Diplococcus carlative Sanguinis

Asobacillus

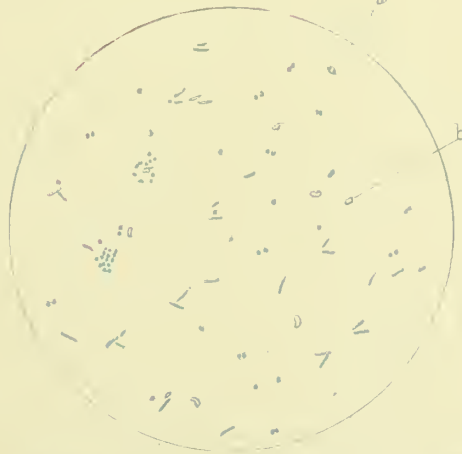
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Bacillus anthracis (spores and vegetative cells)



Bacillus scarlatinae
x 1000.



1000 magnification: tube taken from the skin, but which was already pure culture of *Bacillus scarlatinae*



Bacillus anthracis
after 12 days' growth



Bacillus scarlatinae
after 12 days' growth

are the most frequent carriers of the contagion, though others claim like dangerous properties for mucus, urine, and the fæces. It is certainly true that the contagion of scarlet fever may be carried by almost every conceivable article of apparel or material used about the sick, for next to the variolous microbe the scarlet-fever contagion preserves its vitality for a longer time than any other of the exanthematous poisons. Dr. Holland relates an extraordinary case where the virus survived two generations, being packed away in clothing in a chest for thirty-five years, at the end of which time it communicated the disease to a grandchild for whom some of his grandfather's clothing was made over. To the writer's knowledge, the disease remained hidden in a fur cloak packed away for more than a year, and then communicated the disease to an entire logging community isolated for the winter in the wilds of Northern Michigan. Hence the exact origin of any given case of scarlet fever is often most difficult to accurately settle, especially when we remember the possibility of the disease being carried by books, letters, or toys from some previous case.

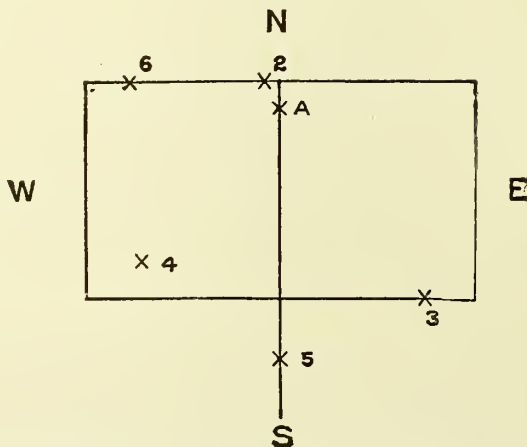
Next to library-books, letters, clothing, and toys, milk seems frequently to be the medium of contagion. In one instance milk is known to have carried scarlatina to one-half of the families to which it had been delivered, although it had not been touched by the milkman or other members of the infected family (Taylor); and in another the disease was carried to all the families served save one, which consisted only of elderly people (Bell). Powers and Klein still teach that the disease originates from the sore teats of infected cattle suffering from bovine fever, but, after much heated discussion on the subject, it appears that the disease thus communicated is modified cow-pox rather than true scarlet fever (Hendon epidemic, 1885). The persistence of the scarlatinal virus in clothing and apartments after ordinary methods of disinfection is sometimes amazing. J. Lewis Smith relates the case of a Sunday-school librarian who contracted the disease from books returned from an infected tenement-house. One month after his recovery the room in which he had been sick and his clothing were disinfected with burning sulphur, and yet he succeeded in carrying the disease personally to his sisters after a journey of three hundred miles to an isolated country town, to which they had been quarantined. These sisters infected the room in which they were confined, so that children visiting it, after its disinfection, in turn contracted the disease. The writer knows of a building in the city of Chicago in which, in three successive years, the children of the families moving into the house contracted scarlet fever in spite of yearly domestic disinfections.

MODE OF TRANSMISSION.—Although it is usually believed that the scarlet-fever poison is not volatile and cannot be carried by the atmosphere solely, the case sketched in the description of Fig. 1, contributed by an intelligent medical student, apparently contradicts previous statements on this subject.

BACTERIOLOGY.—Illingworth still claims, I believe, that the germs of scarlet fever are set free during the fermentation of animal and vegetable refuse. The inhalation of these causes them to lodge upon the mucous membrane of the throat, where they propagate, and, by the reabsorption of their products, produce the other lesions of scarlet fever. Almost all other authorities believe that there is a specific scarlet-fever microbe, which requires a previous human being for its host. Repeated efforts have been made to isolate this micro-organism. As early as 1882, Ecklund of Stockholm thought he had discovered it in the form of colorless discoid corpuscles, about one-tenth the size of the red corpuscle, and found in immense numbers in the urine of scarlatinal patients. These he named *Plox scindens*. He states that he had found them

in vast numbers in the soil and ground-water of the island of Skeppsholm during an epidemic of scarlet fever there. Their presence seems to be well proven, but their relation to scarlet fever is by no means as definite. More

FIG. 1.



"The above rude map shows the relation of, and distance between, several houses in the township of Claremont, Minn., one inch representing a mile. In the house A lived Win. Connell. During February of 1879 one of his children contracted scarlet fever through a letter that came from relatives in Toronto, Canada. About three days later a second child came down with the disease and died on the ninth day. The wind had been blowing from the north-east, and about this time my younger brother came down with the disease in house No. 4. Young James Connell was buried on the day after his death; and on that day the wind changed into the north-west, where it continued for some time. The bedding and clothes of the Connells were hung on the clothes-line to air, and in about one week from that time the children in house No. 3 were taken with the disease. In house No. 2, thirty rods north, there were five children, in house No. 6 there were four children, and in house No. 5, two children. All of these escaped the disease. There was absolutely no communication between the houses on account of the cold weather and fear of the disease. Two years later there came an epidemic of the disease in that vicinity of a severe type, and all the children in the neighborhood had the disease, except those that had had it two years previously."

hopeful are the results of Dr. Edington of Edinburgh, who began in 1886 to make investigations of the blood and epidermis in human scarlet fever. He succeeded in isolating a *diplococcus scarlatinæ sanguinis* and a *bacillus scarlatinæ*. Inoculation of the bacilli produced in rabbits erythema and desquamation; in calves, fever and a rash, followed by desquamation. Dr. Edington says "the bacilli measure 1.2 to 1.4 micro-millimetres in length and 0.4 micro-millimetre in width, and are found in the blood during the first two days only, in the desquamating epidermis only after the twenty-first day, and in the eighteen intermediate days they cannot be demonstrated in any of the tissues." His results have been confirmed by Dr. E. O. Shakespeare, who proposes the provisional name of *bacillus scarlatinæ* for this micro-organism, and reports that, "sown on gelatin-plates, it forms little points of liquefaction after several days. Sown in test-tubes of Koch's jelly, it rapidly liquefies it, but with no distinct growth-formation. The fluid thus formed is crowded with the motile bacilli, but a pellicle is not formed until the liquefaction is well advanced." This occurred in every case but one of the tubes made from the desquamation if taken after the termination of the third week, but never before this. It also occurred in every tube made from scarlatinal blood if taken before the third day of the fever. Inoculation upon rabbits produced erythema, best marked in the old, and in from two to five days a fine desquamation, which lasted for a week to ten days. Temperature, 103°-106° F. Similar results were obtained from guinea-pigs, except that the desquamation was more copious and the hair fell out if pulled upon.

"A calf was then inoculated, and at the same time given some of the

culture in milk. The calf was in good health at the time, and had a temperature of 99.5° F. Six hours from the inoculation the calf developed great sickness, and the temperature taken in the axilla registered 103° F. [This was at 10 P. M.] The calf was then left for the night, but in the morning was found dead. Small portions of the spleen and kidneys were taken from the animal, placed in Koch's jelly, and allowed to incubate, and developed the characteristic bacillus previously described. A second calf was inoculated, when only one day old, with the bacillus, care being taken that the inoculation was made with the absolutely pure material. Previous to the injection the calf's blood was examined, and found to contain no organisms. The inoculation was made in this case with a very carefully sterilized hypodermic syringe. At 6.30 P. M. this was performed, the temperature per rectum then being 99.6° F. At 10 P. M. the animal took milk freely, and the temperature remained practically the same. Next morning, temperature 104° ; sickness, slight diarrhoea, and great prostration, and the throat inflamed. In the afternoon the skin of the thorax, upper abdomen, and inner side of the foreleg presented a general redness, increasing toward evening (T. 102.8°). The next morning the animal was better, but rash still vivid, throat and posterior part of the tongue inflamed (T. 102°). From this time the beast steadily improved, and on the sixth day desquamation set in."

The same bacillus, according to Dr. Shakespeare's report, may be obtained from the blood of a scarlet-fever patient during the first two or three days of the disease, and from the desquamating scales on the twenty-first day in an ordinary case; if malignant, they may be obtained earlier. These bacilli rapidly increase in warm milk, which they may thus infect.

"The rapidity of the growth of this organism—which is such if one inoculate a flask of broth the diameter of which is two inches and a half, and if it be incubated, the pellicle will develop and cover it entirely over in the course of four hours—suggests an explanation of the short incubation of scarlet fever when furnished a proper pabulum."

Such, it seems to the writer, is a fair statement of our present knowledge on the subject, to be confirmed or reversed by later investigations.

Pathology.—Aside from its bacteriology, still in dispute, there cannot be said to be any pathological changes pathognomonic of scarlet fever. Autopsies made upon those dying in the earlier days of the disease show only the local lesion of the throat and engorgement of various internal organs, especially the intestines and brain. Deaths occurring later are generally due to septicæmia or nephritis. The former are apt to show secondary pneumonia and metastatic abscesses, and the blood coagulates poorly and is prone to form clots in the right ventricle. The characteristic changes of pleurisy, pericarditis, endocarditis, purulent meningitis, empyema, or pulmonary gangrene may be found in these cases.

The kidney lesions are those of an acute exudative (Delafield) or glomerulonephritis (Welsh), the latter being the true post-scarlatinal nephritis. In such cases "the liquor sanguinis and the red and white blood-cells escape from the renal vessels into the tubules. Swelling or necrosis of the renal epithelium, with changes in the glomeruli, occurs."

Macroscopically, the kidneys are large and flabby, and the cortex is thick and pale, with injected capillaries. The tubal epithelium is swollen and opaque. Hyaline cylinders identical with the casts are found in the convoluted tubes, and more abundantly in the straight tubes, along with irregular masses formed from the exuded blood-plasma. In the tubes are also red and white blood-cells. The glomeruli exhibit important changes. They become larger or more

opaque, due to the swelling and growth of the cells on and in the capillaries, "for the glomerular capillaries in their normal state are covered on their outside by nucleated cells, and flat cells line their inner surfaces in places, not continuously. On account of these cellular changes, the individual capillaries in the glomerulus become indistinct, but the main divisions of the tufts are visible. In very severe cases the growth of the cells on the tufts is so considerable that they form large masses of cells between the glomerulus and its capsule. The walls of the arteries in the kidneys may be thickened by a swelling of their muscular coats, and the Malpighian bodies may stand out like grains of sand."

This connective-tissue growth Delafield considers characteristic, "involving not the whole of the kidney, but symmetrical strips or wedges in the cortex, which follow the line of the arteries. These wedges are small or large, few or numerous, regular or irregular, in different kidneys, but in every wedge we find the same general characters: one or more arteries, of which the walls are thickened; glomeruli belonging to these arteries, with a large growth of capsule; cells compressing the tufts; a growth of new connective tissue in the stroma around and parallel to the arteries. Between the wedges we find at first only the changes of exudative nephritis; later, a diffuse growth of connective tissue. If the nephritis is of acute type and longer duration, the tissue is denser and has more basement substance. Where the growth of the new tissue is abundant the tubes become small and atrophied. The exudation from the blood-vessels is very considerable, so that the urine contains large quantities of albumin, many casts, and red and white blood-cells" (Delafield and Prudden). The irregular distribution of these kidney lesions, according to Bartel, explains the contradictory results often obtained by successive examinations of the urine. There may be parts of the kidney which entirely retain their functions, and from these normal urine may be secreted. But that a scarlatinal dropsy may exist from beginning to end without the presence, at any time, in the urine of either blood, albumin, or casts, is as improbable as that dropsy may occur without nephritis (Bohn).

Incubation.—Formerly a week or ten days was given as the usual length of the stage of incubation; later writers, however, fix it at two to five days, and it may, in malignant cases, last not more than twenty-four hours. But it is often difficult to say exactly when the stage of incubation ends and that of the initial sore throat begins. Murchison's table (Smith, p. 275) shows that in the great majority of the cases reported by him the stage of incubation was within five days, and the latest writer on this subject says that if the initial vomiting be taken as the conclusion of the stage of incubation, it will be found to be under three days (Ashby, p. 248).

Symptoms.—The onset of scarlatina is usually so abrupt that its beginning may be fixed with considerable definiteness. There is possibly a previous slight duskiness of the skin, chilliness and malaise, but usually the first thing that attracts attention is vomiting, often without any relation to a previous meal; or there may be diarrhœa. Older children may not actually vomit, but complain of nausea, languor, headache, and sore throat, and feel chilly, although the face is flushed, and the thermometer may show a temperature as high as 103°–105° F. If such children are also drowsy, they may become delirious in their sleep. The pulse is full and strong (120–160), the skin is hot and dry, and the throat feels stiff and uncomfortable, and, if examined, will show a characteristic punctate redness. Such is the ordinary onset of a typical case of scarlet fever, but there is no disease of childhood that is liable to wider and more eccentric variations in its onset and course,

PLATE VII.

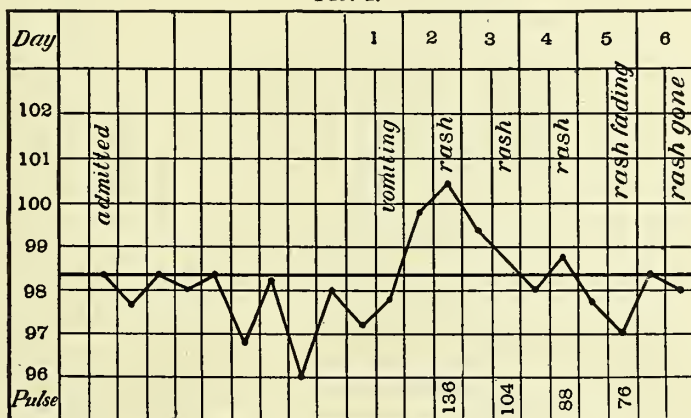


SCARLET FEVER.

oscillating between the very slight abortive form and that frightful variety called by the French *foudroyant*, or scarlatina fulminans, fortunately rarely met with; for in such cases the child succumbs, mortally poisoned from the very first by the virulence of the scarlatinal virus, without any prodromal stage or hardly any symptoms except those which may be referred to the nervous system. These dreadful cases often run their entire course in from thirty-six to forty-eight hours without eruption or sore throat, the only symptoms being nausea, dizziness, loss of consciousness, coma, violent delirium, or convulsions attended with abnormally high temperature (107°).

Scarlatina simplex may be differentiated in twenty-four hours by the appearance of the typical scarlatinal rash in the form of a scarcely perceptible scarlet flush or pin-point eruption, very closely resembling in color and stippling the shell of a freshly-boiled lobster. The eruption usually begins on the neck or cheeks or small of the back, and ought in forty-eight hours to spread nearly over the body, either as a well-defined blush or in scarlet patches—*scarlatina levigata*. Plethoric and blond children develop the rash most promptly, and in all cases its color is heightened by the warmth of the bed, by hot baths, or by crying. A characteristic white line remains for a few seconds after drawing the edge of the nail or the point of a pencil over the rash. This typical line is supposed to be due to a paralysis of the vaso-motor

FIG. 1.



Temperature Chart in a Mild Case of Scarlatina. Patient 6 yrs. old. (After Ashby.)

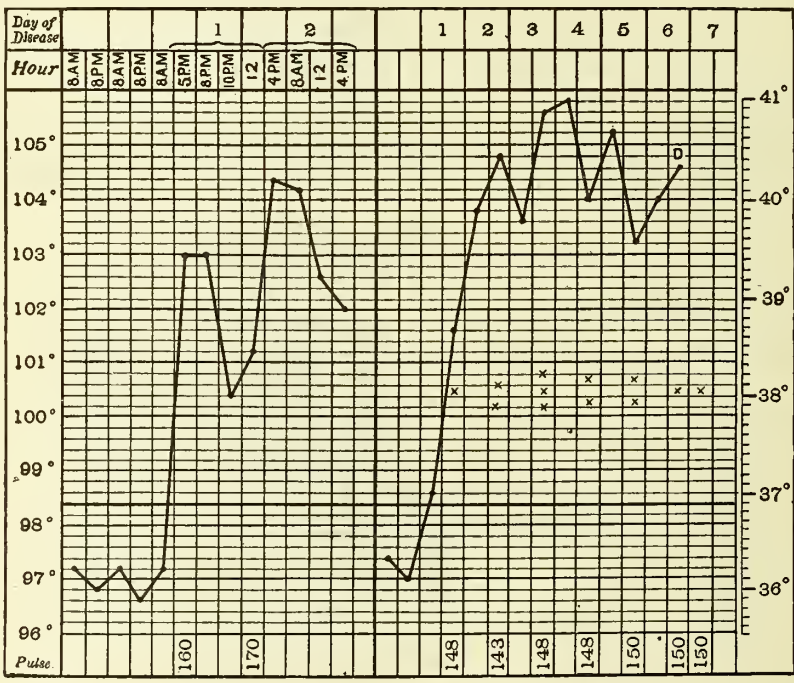
nerves of the capillaries in these congestive areas. Until the eruption is well marked the fever continues high, often dangerously so, as it is not unusual to find the temperature in impressible children marking 105° – 107° F. The pulse is quick and sthenic, except in cases of scarlatina maligna, where there may be general depression, delirium, and collapse from the very onset of the disease. The pulse, as a rule, is faster than the temperature would apparently call for, ranging from 130–150, its relation to the rash and temperature being well shown in the accompanying chart, taken from Ashby (Fig. 1). Pharyngitis, with more or less soreness of the throat, is always present, although it may not be sufficiently painful to cause the child to complain (*scarlatina sine angina*). The respiratory organs, except the throat, are rarely involved, so that cough is generally absent. When present, it is due to faucial irritation, except where pneumonia occurs later as a dangerous complication. The tongue is the so-called strawberry tongue—that is, covered with a white fur with bright red tip and borders.

When the papillæ are greatly swollen, they cause the granular appearance known as the raspberry tongue. Some writers speak of a pathognomonic sweetish odor of the breath which may be detected at this time, but this is by no means an invariable symptom nor one upon which much reliance should be placed.

In a simple, uncomplicated case the fever and all threatening symptoms moderate with the appearance of the rash, with the exception of a slight evening febrile exacerbation, and any variation from this rule betokens malignancy or some new complication.

From the fourth to the sixth day desquamation ordinarily begins. Those areas which are first reddened fade in like order, and, as the color disappears,

FIG. 2.



Temperature Chart of Malignant Scarlet Fever. Death in 24 hrs. (After Ashby.) | Temperature Chart of Malignant Scarlet Fever. Death on 7th day. Rash indicated by *.

the skin is found to be covered with loose branny scales. These scales drop off imperceptibly, except when from itching, as is apt to happen on the face and neck, they are scratched off, and the tender epidermis beneath becomes cracked. In such cases the scales may be thrown off in shreds, or casts of the entire lip, fingers, or palms of the hand may be shed. A like desquamation occurs from the membranes of the throat, trachea, kidneys, and intestines, though of course the epithelial scales in these localities are carried away in a softened, macerated condition.

Out of 200 cases reported, 11 reached their highest temperature on the first day, 76 on the second, 75 on the third, 36 on the fourth, and only 2 on the fifth day. When the highest temperature is reached after the fifth day, or if the temperature has not fallen considerably by that time, some complication is certainly keeping it up, so that the thermometer and violence of the nervous

symptoms form a valuable criterion as to the danger of the child. A dull, apathetic condition is, as a rule, more to be dreaded than the usual restlessness, which is due to continued reflex irritation of the rash. In hyperæsthetic children this produces twitching, or even eclampsia, which is graver the later it occurs in the disease.

Variations.—We have previously described what might be considered a typical case of uncomplicated scarlet fever, but, unfortunately, uncomplicated cases are so rare that there is no disease of wider variations in every symptom.

The *eruption* may be so light as to escape detection, or, on the other hand, instead of the ordinary scarlatina lævigata, the eruption may appear in the form of small nodules (*scarlatina papulosa*), in which the papillæ of the skin are swollen, and the whole body looks as if covered with goose-skin. Or, again, these papillæ may become covered with vesicles, and we have that form of scarlatina which is known as *scarlatina miliaria*. Should these vesicles become merged together, they give an eruption to which the name of *scarlatina pemphigoides* seu *bullosa* is given. Such variations are found most frequently on the face, and are usually of grave import. Vogel reports exceptional cases in which the eruption was intermittent in character, appearing only at certain times of the day, and for this he proposes the name of *scarlatina intermittens*. Lastly, we may find that fatal form to which the name of *scarlatina petechialis* seu *hæmorrhagica* has been given, where there is an actual extravasation of blood into the skin, and hence the popular name of "black scarlet fever" by which it is sometimes known. In nervous children it is not infrequent to find urticaria accompanying scarlet fever, masking the characteristic rash. Vogel also reports a curious variation of scarlatinal rash in which are found sharply-marked, isolated areas which remain milk-white in color, or at least much whiter than normal integument, due to a temporary paralysis of the arterioles similar in character to that which follows the thumb-nail mark on the normal scarlatinal flush; but they are more persistent in character and are usually of unfavorable portent. Any intercurrent disease, as enterocolitis, which produces a determination of blood from the surface of the body, may greatly delay the appearance of the rash or render it so light that its differentiation will be difficult.

Complications.—*Throat.*—The angina of scarlet fever may assume any form, from simple catarrhal injection to extensive necrotic destruction of tissue. Ordinarily, a bright red flush, with punctate marks, such as might have been produced by a small brush dipped in red ink and dotted over the pillars of the fauces, is the earliest and one of the most characteristic symptoms of scarlatina. This may proceed no further than to give slight difficulty in swallowing and to impart a nasal tone to the voice. But, on the other hand, and more frequently—especially if pharyngeal disinfection is not practised from the very first—the swelling becomes so great as to make swallowing almost impossible. In such cases fibrinous exudates appear on the tonsils and fauces, and should the inflammation not be limited to the palate and fauces, the exudate may extend into the post-nasal cavities, the larynx, and even into the œsophagus and stomach. More frequently it proceeds through the Eustachian tube into the internal ear. (See Otitis Media.) The differentiation between the fibrinous exudate of scarlatina and true diphtheritic membrane is by no means easy, the more so since undoubtedly true diphtheria is not infrequently grafted upon the necrosis of scarlatinal angina; but it may be helpful to remember that the exudate of scarlatina is yellowish and pultaceous, rather than the ashy-gray membrane of true diphtheria. Should the presence of Loeffler's bacillus

be finally accepted as pathognomonic of diphtheria, the differentiation may then be made absolutely; whereas at present we must frequently remain in doubt, since the removal of the scarlatinal exudate leaves the superficial layers of the pharyngeal mucous membrane denuded and bleeding exactly as in diphtheria. A similar gangrenous process may proceed upward into the pharynx or along the Eustachian tube into the cavity of the middle ear, with all the perils of purulent meningitis which this implies. Similarly, as in true diphtheria, the exudate may pass downward into the larynx, where its presence is made known by a characteristic croupy metallic cough. If the exudate attacks the nasal cavities, this is attended by a profuse excoriating discharge, which soon grows purulent and offensive in odor.

Adenitis.—All forms of scarlet fever are attended with inflammation of the lymphatic glands of the neck, and, as a rule, it will be found that the involvement of these glands bears a direct relation to the severity of the throat lesions. So we find all grades of adenitis, from the slight induration which may be found accompanying all varieties of scarlatina, to a brawny swelling of the glands and cellular tissue embracing the whole neck. Such extensive mischief betokens like serious necrotic processes taking place within the pharynx, where the poisonous débris clogs and inflames the lymphatic glands, their pressure and morbid processes inflaming contiguous tissues. This cellulitis may extend from ear to ear, until deglutition becomes difficult and wide opening of the mouth impossible. If relief does not come early by resolution, the widely-distended tissue gives way to suppuration or gangrene, and death from hæmorrhage or septicæmia occurs.

Scarlatinal Arthritis is not infrequently met with in certain epidemics of scarlet fever during both the eruptive and the desquamative stage. This form of arthritis attacks by preference the knee- and elbow-joints, and scarcely can be distinguished by its objective symptoms from ordinary articular rheumatism, being, like it, excessively painful. But arthritis rheumatica rarely ends in pyæmia or permanent articular osteitis, as arthritis scarlatinæ is very prone to do.

Diarrhœa and Dysentery are not at all infrequent complications after the crisis of the disease, probably being caused by desquamation of the intestinal epithelium, analogous to that which undoubtedly occurs in the tubuli uriniferi at this time.

Scarlatinal Nephritis.—Last and, justly, the most dreaded of the complications of scarlatina, is that form of nephritis which so frequently occurs during the course of the disease that it may almost be considered pathognomonic; for a mild grade of renal catarrh is as constantly present as is desquamation (Steiner). It is true this frequently escapes observation and passes on to recovery without special treatment, but its existence is always a potential cause of morbus Brightii scarlatinus, which should be considered not as a distinct disease, but as an intensification of the previous catarrh of the tubules brought about by chilling of the skin, etc. (Bohn).

Similar nephritic catarrh has been noted in measles, small-pox, pneumonia, and other diseases, induced, as the writer believes, by the passage through the kidneys of irritating ptomaines generated in the body by the specific microbes of these diseases. The excretion of these or analogous compounds through the skin very likely gives rise to the characteristic rash, hence analogous lesions might be inferred for the kidneys. It is a well-known fact that the lighter the cutaneous rash the more liable are the kidneys to be seriously implicated, presumably from increased excretion of various ptomaines through organs now endeavoring to do the work of both skin and kidneys. Daily examination of

the urine should be made for at least two weeks in even the mildest cases of scarlet fever, and will show from the beginning of the eruption evidence of renal catarrh (epithelial debris and albumin), although the kidneys are apparently working normally. While the urine is high-colored and deposits copious urates, Dr. Gee claims that urea is not necessarily diminished. The chloride of sodium is lessened until the fourth to the sixth day, and phosphoric acid after crisis; while the urates or uric acid appear to excess during convalescence. In other cases the urine is cloudy, and contains fatty renal epithelia, more rarely hyaline casts, and red and white blood-corpuscles (only exceptionally albumin), all of which disappear usually with the disappearance of the eruption, but may progress to an actual catarrhal nephritis. This renal catarrh Bartel believes is due to a specific poison—ptomaine (?)—circulating in the blood, which poison irritates the tubules of the kidneys in its passage through the Malpighian tufts, either directly or from irritating properties imparted to the urine before its percolation through the tubuli uriniferi. Others claim that the source of this irritation lies in certain specific micrococci circulating in the blood, being analogous to diphtheritic nephritis, which Oertel thinks due to bacterial emboli.

A diminution in the quantity of the urine is often the first thing that awakens the attention of the physician, if he makes it his duty, as he ought, to keep himself posted daily until the end of the third week. The normal amount of 800 to 900 c.c. per diem may fall suddenly to 100 or 50 c.c., or even less. Its color is yellowish-red, sometimes almost yellowish-green when cooled; turbid, or clearing up on standing, depositing a cloudy precipitate made up of kidney cells and casts, urates, and uric-acid crystals in varying proportion. At times the urine is blood-red or smoky brown, from the blood it contains. Under the microscope the precipitate is found to consist of varying quantities of kidney epithelia, partly normal and partly swollen and distended, cloudy, and undergoing fatty degeneration. Besides these there may be various forms and phases of casts, lymph-corpuscles, red blood-corpuscles, and the crystals of urate of sodium and uric acid. The quantity of albumin found in urine is deceptive, since in certain epidemics of scarlatina, even where dropsy suddenly appears, often only faint traces of albumin may be found in the urine. Or albumin may be entirely absent during certain times in the day, or even for several days at a time, or during the greater part of the disease. Or, again, unmistakable albuminuria may be present while the urine is clear and free from all other abnormal elements. It may even happen that frequent analysis of the urine for days may fail to show either casts, epithelial cells, or crystals, while all of these, together with albumin, may be found at a subsequent examination.

Scarlatinal dropsy is often the first warning of the existence of any kidney lesion in mild cases which are supposed by parents, and even by the physician, to be well along in convalescence. As a rule, the chief danger of scarlatinal nephritis lies about the end of the second week or during desquamation, though dropsy may appear as late as the fifth or sixth week. The first symptoms noticed are slight cedema of the face and swelling of the eyelids. These are followed by puffiness of the backs of the hands and feet, sometimes unilateral, with dropsical enlargement of the abdomen. In the case of children who have not yet been allowed to rise from their beds the anasarca is often most marked in the back and in the genitals, which may become frightfully swollen and sensitive. As a rule, the kidney complication is ushered in with a return of fever, or an increase in fever, if it still be present. But there is also a feverless nephritis, without subjective symptoms, loss of appetite, or anything abnormal that can be detected. In other cases there is only an evening

increase of temperature and pulse. Generally the skin is dry and ceases to desquamate. Pain over the kidneys is seldom complained of, unless questioned about or obtained by pressure. If the disease in the kidney is limited, there may be only a localized œdema, such as hydrothorax, hydrops pericardii, œdema of the lungs, or dropsical effusions into joints. This localized œdema may follow a brief apparent convalescence, during which children recover their appetite, and exhibit no features of illness, unless it be the persistence of slight lassitude and fever at night. After exposure to cold such cases develop anorexia, depression, and pain over one or both kidneys. The amount of urine is greatly diminished. It is concentrated, high-colored, and contains albumin and casts, and may not measure more than an ounce for the entire day, or may even be completely suppressed. About 6 per cent. of all scarlatina patients suffer from post-scarlatinal nephritis, the course and duration of which depend directly upon the extent of the anatomical lesions of the kidney. Very light cases recover in a few days. Generally the anasarca and effusions increase for several days—say a week and over—breathing being hindered by the ascites and pleural effusions, and the nights are restless. Œdema of the lungs provokes incessant coughing. Swelling of the genitals is often painful, but does not noticeably interfere with urination. Death may ensue suddenly from uræmic convulsions when danger is least expected. Ashby attempts—and it seems wisely to the writer—to differentiate between septic and post-scarlatinal nephritis, either of which may be met with during the course of scarlet fever. The urine in the first contains no blood-corpuscles, but is highly albuminous, and is not attended with dropsy nor uræmic convulsions. Autopsy in these cases shows a distinctly softened, pyæmic kidney, which contains minute abscesses, and is mottled in its cortex with injected blood-vessels and inspissated pus. Death occurs from pyæmia, and not directly from the kidney lesions, which are only a part of the more general process. In the second class of cases death results from uræmia. The lesions of the post-scarlatinal kidney have been fully described under Pathology.

Sequelæ.—*Chronic nasal catarrh*, *ozæna*, *pharyngitis*, or *hypertrophy* of the tonsils, with acute attacks of *quinsy*, or *suppurative otitis*, with *chronic otorrhœa* and *deafness*, more or less complete, are among the dreaded reminders left after scarlatina, especially where the *angina* has been malignant. In many such cases the tonsils become deeply excavated, and the soft palate sloughs; but even under these circumstances recovery is possible. Or, as has previously been noted, *diphtheritic-like membrane* may cover the fauces, palate, and even spread on to the *epiglottis* and into the *larynx*. Death from exhaustion or *hæmorrhage* usually terminates such cases, or, if life is for a while prolonged, death comes later from *septicæmia*, often terminated by *septic pneumonia* (seventh to fourteenth day). But even *septic pneumonia* is not necessarily fatal, for recovery took place in one of the writer's cases after the appearance of this sequel subsequent to *otorrhœa* and *cervical abscesses* and *sloughing*. The amount of damage sometimes inflicted by these *cervical sloughs* is frightful. Smith speaks of one which laid bare the *carotid* and produced death by its perforation. Williams relates a still more remarkable case, in which *superficial ulceration* of the fauces, palate, and tongue was conjoined with *suppuration* of the *lymphatics* of the neck. This was followed by *sloughing*, exposing, in the triangle of the neck, a space bounded by the edge of the *sterno-mastoid*, the upper border of the *thyroid cartilage*, and the median line of the neck. Nevertheless, under *antiseptic treatment*, the boy made a good recovery, although he was only six years of age and had previously been considered delicate.

Broncho-pneumonia, *pleuro-pneumonia*, *empyema*, and *peritonitis* are among

the possible complications of scarlatinal nephritis. If the temperature runs high, the tongue becomes dry and brown, the urine scanty and albuminous, and death rapidly ensues. But milder cases are not hopeless if the urinary secretion can be re-established.

Cardiac dilatation, endocarditis, and pericarditis are the more frequent heart-lesions that should be guarded against in every scarlatinal nephritis, for, conjoined with increased arterial tension and general malnutrition, they may bring sudden death either from heart failure or embolism. The possibility of such untoward termination to nephritis should never be forgotten, for no sharper reproach can come to the physician than the thought that had he allowed less work to be thrown upon a weakened heart he might have carried his patient into safe convalescence.

Otitis, with perforation of the membrane, more than any other sequela, has too often been left a lifelong reminder of scarlet fever. In many of these cases little pain is complained of, although the fever remains suspiciously high until a purulent discharge from the ear makes its appearance. Mastoiditis or purulent meningitis may prove fatal, but in a majority of these cases no such complications take place, and the child recovers, more or less deaf or afflicted with a chronic otorrhœa. According to Batut, statistics in Belgium show that out of 1892 cases of deafness, 216 followed scarlet fever. Another observer found out of 400 cases 144 due to the same cause.

Synovitis has already been referred to under the head of Arthritis, as liable to occur about the second week. Suppuration and pyæmia are the chief dangers in these cases.

Cerebral lesions, such as paralyses, blindness, aphasia, loss of memory, hemiplegia, etc., are among the sad sequelæ of the uræmic convulsions of scarlatinal nephritis.

Convalescence from severe cases of scarlatina is always protracted, the subsequent anæmia lasting for months or years, especially in scrofulous children, in whom the virulence of the poison is most lasting in its effects. Many of the most discouraging cases that come into the hands of the physician dealing largely with the diseases of children are those in which the child's vitality has been undermined by malignant scarlatina. Such children frequently suffer for years from the so-called mucous disease of Eustace Smith or from renal incompetence. In other cases there is a chronic otorrhœa or offensive ozæna, which renders their lives miserable, and so saps their vitality that they succumb easily to intercurrent disease. This is especially true of those children in whom the functions of the kidneys have been seriously crippled by post-scarlatinal nephritis. Such a previous history always awakens serious apprehensions in the presence of diphtheria, typhoid, or any septic disease.

Diagnosis.—The early diagnosis of a mild case of scarlet fever is often a matter of great difficulty, but it is a matter of no little importance to the patient, for such mild cases seem to be the ones most liable to nephritic complications. Since mild cases may communicate dangerous attacks to those more susceptible, it is always safe to give the well children the benefit of your doubt by isolating all suspicious cases. Nausea, pain in swallowing, and fever constitute a trio of symptoms sufficient to isolate a patient until a rash of some kind appears. This may be so light and transient, especially if there be coincident diarrhœa, that it may escape detection unless carefully watched for; and even then there is an *erythema scarlatiniforme* that without previous history may deceive the very elect in pædiatrics. In such cases, however, the throat does not show the characteristic stippling of scarlet fever, and a brisk emetic or purge brings the case to a speedy termination. The early differentiation of

rubella from scarlatina is often puzzling, but Jamieson calls attention to the fact that in rubella the characteristic tongue of scarlet fever is absent, while the mild catarrhal symptoms of the former are not ordinarily present in the latter disease.

The eruption of measles is most distinctly patchy, and is preceded by several days of drowsiness and the symptoms of an ordinary cold. But in all doubtful cases isolate and wait for light, remembering "that nephritis occurring after an anomalous rash makes it practically certain the primary attack was scarlet fever." Broncho-pneumonia under similar circumstances justifies a diagnosis of measles.

Prognosis in scarlet fever must be largely influenced by the character of the then prevailing epidemic and the general condition of the child. The virulence of the scarlatinal poison and the susceptibility of the one attacked determine the degree of restlessness, jactitation, and delirium observed. Initial eclamptic attacks rarely occur, except in unusually nervous, susceptible children, and their occurrence is of very unfavorable portent.

As a rule, the early and extensive implication of the cervical lymphatics is a forerunner of serious throat complications. Nasal diphtheria complicating scarlatina is of the gravest import, and the gravity is proportionate to the early age of the child, children under four years giving as high a mortality as 28 per cent. The younger the child the more guarded should be the prognosis, especially when associated with diarrhœa, which is regarded by Ashby as a very serious symptom.

Where the temperature continues high (104° – 106°), and there is much diarrhœa or extreme restlessness, or the angina is malignant, the prognosis is always grave. Drowsiness is always an unfavorable symptom, and a high temperature continued into the second week is sufficient ground for anxiety.

Desquamation is seldom completed before the sixth week, and is not always at an end in twice that time, Finlayson fixing the infective period of this disease as seven to eight weeks.

The nephritis complicating or following scarlet fever is more dangerous than the primary disease. Where persistent vomiting occurs, not only on the first, but on subsequent days, the prognosis is correspondingly grave.

Post-scarlatinal nephritis is the most favorable form of parenchymatous inflammation of the kidneys, usually ending in recovery in two or three weeks by means of copious diuresis, but it is worth remembering that the excessive excretion of uric acid, which persists well into convalescence, may form gravel or calculi. As a rule, epithelial casts and detritus persist after the disappearance of the albuminuria, sometimes for an exceedingly long time, especially in cachectic children.

Death rarely occurs before the fourth day, and usually not later than the seventh, except from post-scarlatinal nephritis. Sudden death may result from rapid and uncontrollable increase of dropsy, either into the peritoneum, pleura, pericardium, or ventricles of the brain, or from œdema of the lungs or glottis. Or, stopping short of immediately fatal results from œdema, the end may come more slowly from inflammation of the lungs or pericardium, or still more slowly from gangrene of the genitals or from bed-sores. Or, as may be inferred from the above, the nephritis may assume a chronic form.

The relation between the intensity of the scarlatinal eruption and the danger of subsequent nephritis is by no means constant, although the writer has come to dread its appearance in the lighter cases because these are the ones in which the care of the parents is apt to be relaxed with the apparent rapid convalescence of the child.

Serious cerebral affections, such as paralysis, blindness, aphasia, loss of memory, hemiplegia, may remain as sequelæ of scarlatina.

Mortality varies widely with the epidemic. That in the Manchester Children's Hospital varied from 6 to 25 per cent. according to the epidemic, the average for ten years (1877-87) being 11.8 per cent. Of 10,000 cases reported by Collie, the mortality was 12.5 per cent. for all ages, that between three and four years reaching as high as 25 per cent.

These figures, it must be confessed, are too high for the average American practitioner, but he may, like foreign physicians, be compelled to radically change his ideas on the subject. Brettonneau, for instance, up to 1799 thought scarlatina the mildest of all the exanthemata; and so also the Irish physicians thought from 1804 to 1831. But Brettonneau was obliged to entirely change his views after encountering the fatal epidemic at Tours in 1824; and a similar outbreak in Dublin in 1831 completely revolutionized the views of the Irish physicians in regard to the fatality of scarlet fever.

Treatment.—A hopeful fact, always to be borne in mind in any choice of treatment adopted in scarlatina, is that it is a self-limited disease, and that no remedy has yet been discovered that will either abort or greatly modify its course. The medical literature of the past twenty-five years teems with alleged specifics, but all of these by subsequent trials have been found no better nor worse than those proposed before them. Nevertheless, the intelligent physician owes it to himself and his patients that he shall not desert them upon the rocks of medical agnosticism nor wreck them upon the snags of polypharmacy. If he cannot abort the disease, he may make its course less uncomfortable to his patient, and by careful foresight ward off many a threatening complication.

DIET is not unimportant in scarlet fever, for our aim from the very beginning should be to tax the kidneys, already in a catarrhal condition, as little as possible with nitrogenous materials. Hence the ideal food for the scarlet-fever patient is koumyss, skimmed milk, or milk and Vichy. But the ordinary American child will not long tolerate such light diet, especially when rapidly convalescing, so we are usually forced to add to our diet-list broths, soups, light puddings, and baked apples, happy if thereby we reduce meats to a minimum. While the writer cannot agree with Jaccoud that a milk diet is an absolute safeguard against post-scarlatinal nephritis, it is true that a liquid diet and warmth should be carefully secured for at least four weeks.

GENERAL TREATMENT.—If the initial nausea is vexatious, it may often be allayed by:

R̄. Aquæ cinnamomi
 'Liquor calcis āā f ʒj.
 Tinct. gelsemii f ʒss.—M.

Sig. Teaspoonful every hour.

For the high arterial tension and fever, tincture of aconite, given according to the plan of Ringer—*i. e.* a drop every quarter hour until arterial tension is decreased, and then given sufficiently to hold the pulse at that point every two or three hours—is very satisfactory.

Chloral hydrate is a favorite with the writer, almost entirely displacing the tinct. ferri chloridi of his earlier practice, except in those cases where there is malignant angina from the beginning. In such cases nothing has been found superior to the tincture of the chloride of iron (one drop per dose for each year of the child's age), with whiskey or brandy, given according to Dr. Chapman's plan. The surprising tolerance of such children for alcoholic stimulants

shows that their power is expended otherwise than in their usual effects upon the brain. Many such children will take fʒss of brandy every hour without showing any of the usual physiological effects. In ordinary cases, however, small doses of chloral hydrate seem to be all that is necessary to relieve restlessness, moderate the angina, and, to a limited degree, act as an antiseptic. For the first forty-eight hours such a prescription as the following has often proven most useful:

R. Chloral hydrate ʒss-j.
 Camphor water fʒss.
 Syrup of orange-peel fʒiss.—M.
 Sig. To alternate with aconite as required.

When the eruption is tardy in appearing, a hot salt or mustard bath will expedite matters, or, if these are ineffectual, packing in a sheet wrung out of hot water and sprinkled with mustard rarely fails.

The throat is too often neglected, and yet here is the focus from which spread many of the dangerous complications of this disease. Local antiseptics may be a modern device, but Underwood came very near to the writer's ideas when he wrote on this subject many years ago: "The throat must be often syringed with . . . though the quality is perhaps of far less importance than its being frequently made use of, which is absolutely necessary, especially in young children. . . . Even syringing the throat with hot water is found to administer immediate relief." The local treatment of the throat with peroxide of hydrogen spray, as directed under the head of Prophylaxis, can hardly begin too early, and the same may be said of the inunction of the body with some antiseptic ointment. Quinine internally may be added later if there is evidence of failing strength.

Cerebral symptoms, unless associated with scanty urine, may be rendered tolerable by the addition of bromide of potassium (grs. v-x) to each dose of the chloral hydrate mixture, with a mercurial purge and the application of cold to the head. Phenacetin is sometimes a great comfort in such cases, but the writer discourages the use of the other antipyretics in scarlet fever, except as a last resort in abnormally high temperature. Even in these cases persistent sponging with cool water, or even cold affusion, ought first to be tried. Persistent drowsiness always awakes suspicion as to post-nasal complications, and emphasizes the necessity of nasal irrigation, frequently repeated.

Scarlatinal arthritis in cachectic children may proceed to suppuration and destruction of the joints, but, fortunately, most of these cases are more painful than dangerous, and yield promptly, like true rheumatism, to fair doses of salicin and codeine and wrapping the affected joints liberally with cotton batting.

Cervical adenitis is more frequently overtreated than neglected, for the swollen and tender glands apparently require immediate attention. And yet the trouble lies farther back, for the débris that blocks these inflamed glands comes usually from the pharynx. Hence efficient pharyngeal and nasal cleansing will do more for adenitis than poultices, lotions, or ointments. So-called energetic treatment too often precipitates the very troubles we are seeking to guard against. Instead of poultices and iodine, simple rest and warmth will often work wonders even in brawny, swollen necks where suppuration appears inevitable. At all events, camphorated oil, applied on absorbent cotton, should be tried before proceeding to more vigorous measures.

Diarrhœa is apt to be quite persistent, and occasionally painful, when once

it makes its appearance. So far, I have rarely seen it assume a dangerous aspect, for it usually can be held in check with paregoric alone or conjoined with bismuth in an emulsion.

SCARLATINAL NEPHRITIS.—Individuals and epidemics of scarlet fever vary so greatly in their liability to nephritis that it is difficult to rightly estimate its prophylactic treatment. From 60 to 70 is given by various authors as the average percentage in dangerous epidemics, and from this it falls to 6 or 7 per cent. in ordinary cases. The writer believes that this latter proportion can be still further reduced by the proper care of children in the mildest form of the disease, for these are the very ones which give us the highest proportion of fatal cases of nephritis. It follows, then, that all children ill with scarlet fever should be kept in bed during the rash, no matter how mild it may be; and, furthermore, such children should be confined to warm rooms, or, better still, to bed, for four or six weeks from the appearance of the initial symptoms. At least twice a week during this time the urine should be examined, and upon the appearance of the slightest unfavorable symptom the child should be sent back to bed again if he has already been allowed to be about the room.

But should these premonitory symptoms be disregarded, or if, in spite of these precautions, scanty albuminous urine and dropsical effusions appear, then the physician's most energetic efforts must be directed toward making the skin or intestines temporarily assume, as far as possible, the functions of the kidneys, throwing on the latter, at the same time, as little work as possible in the way of the excretion of nitrogenous refuse. (See Diet.) The copious use of water, if tolerated by the stomach, will act as one of the very best of the diuretics. Long ago Roberts placed pure spring water at the head of the list, and the writer has not yet found any diuretic to displace it, though lemon-juice, raspberry vinegar, or skimmed milk may be added without harm to induce the child to drink more freely of the water.

Should the urine still remain scanty, then *diaphoresis* must be induced in order to increase the action of the skin—first, by means of baths, and then, if necessary, by drugs. A warm bath (98°–100° F.) for fifteen to twenty minutes is often grateful to the child, and, if supplemented by a flannel pack, is very efficient. The hot-air or steam bath, as described under the treatment of Acute Nephritis, may likewise be employed with success. Any of these methods will be assisted by the internal use of diaphoretics, chief of which are the preparations of jaborandi. Sips of a hot infusion of the leaves (ʒj to Oj) act both as a powerful diaphoretic and sialagogue. To avoid the latter action Smith prefers the alkaloid pilocarpine, $\frac{1}{40}$ to $\frac{1}{20}$ grain, conjoined with an alcoholic stimulant every four to six hours. Should this fail, the same writer speaks highly of the following prescription:

Ry. Potassii acetatis
 Potassii bicarbonatis
 Potassii citratis āā ʒij.
 Infus. tritici repentis fʒviij.—M.

Sig. Teaspoonful every three or four hours to a child of five years.

More palatable and fairly efficient is the following:

Ry. Liq. ammonii acetatis
 Syr. acidi citrici āā fʒij.—M.
 Sig. Teaspoonful every hour in hot lemonade.

Or, where there is considerable dropsical effusion, this can be with advantage alternated with diuretin (gr. j-iv), given in a large amount of water.

Dropsy usually requires, in addition, the free use of some hydragogue cathartic, of which the compound jalap powder (gr. v-x) is certainly the most efficient and unpleasant. Hence, when it is found impracticable to repeat the dose as often as required, it may be supplemented by a cream-of-tartar lemonade, made by dissolving a tablespoonful of the salt in hot water, diluting with an equal amount of cold, sweetening to taste, and adding sufficient claret or port to make agreeable. Most children will take this laxative readily.

Or the following prescription of J. Lewis Smith may be employed:

R \bar{y} . Ol. cinnamomi gtt. viij.
Magnesii sulphatis \bar{z} j.
Potassii bitartratis \bar{z} ij.—M.

Sig. One teaspoonful repeated from two to four hours, until catharsis occurs.

But the use of laxatives should be continued no longer than is strictly necessary, for their repetition brings anæmia, a result greatly to be deplored.

After relieving the initial congestion of the kidneys, stimulating diuretics are helpful; and of these digitalis has justly a high reputation. The infusion is a reliable preparation, and may be given in connection with acetate of potassium, as in the following mixture:

R \bar{y} . Potassii acetatis \bar{z} ss.
Infus. digitalis f \bar{z} vj.—M.

Sig. One teaspoonful every four hours.

Local treatment will also greatly help in relieving the fever and backache. Foreign writers speak highly of the use of leeches over the kidneys in these cases, but the majority of American physicians are willing to rely upon the use of poultices or plasters. A large warm flaxseed poultice, containing mustard or digitalis, often acts like a charm. Smith prefers one made of 1 part each of powdered mustard and ginger to 16 of ground flaxseed, and advises dry cupping when the child is not frightened thereby. Sluggish kidneys may be gently stimulated by caprine plasters or some mildly stimulating embrocation, and a flannel bandage worn day and night.

It ought never to be forgotten that while the liability to heart failure is not as great in scarlatinal nephritis as it is in the convalescence of diphtheria, yet it is a possible danger, and one from which death may rapidly occur. An irregular, flickering pulse requires absolute confinement to bed and the continued use of some chalybeate tonic. A pleasant one may be found in the following:

R \bar{y} . Tinct. ferri chloridi f \bar{z} ij.
Acidi phosphorici dil. f \bar{z} vj.
Glycerini f \bar{z} vij.
Vini xerici f \bar{z} iv.—M.

Sig. Teaspoonful four times a day.

Hæmaturia can best be controlled by gallic acid and ergotine, and threatening convulsions kept in check by rectal injection of chloral and bromide of potassium (gr. v and gr. x) in milk or water. Nitro-glycerine tablets ($\frac{1}{100}$ gr.)

are very valuable for temporary stimulation of the heart, and may be used hypodermatically if the need be pressing.

Prophylaxis.—All attempts to procure personal immunity by means of inoculation have up to the present time proved ineffectual. The same may be said of prophylactic medicaments, for it is more than doubtful whether any known drug has the power to prevent the occurrence, or to greatly modify the course, of scarlet fever after its incubation. Even Hahnemann's vaunted specific, belladonna, has failed so often and completely that it need only be mentioned as one of the curious delusions of medical history. The same may be said of sulphocarbolate of soda (Beebe's), quinine, salicylate of sodium, and the other alleged preventives which from time to time appear and disappear in medical literature. The fact is that epidemics of scarlatina vary widely in their intensity and danger. Hence it is that in one epidemic the liability to contagion is reduced to a minimum, and whatever may be used at that time receives credit for prophylactic powers which fail miserably when next put to the test. Our efforts must, therefore, be confined to isolation of the patient and disinfection of whatever touches or comes from him, for it must be remembered that not only the desquamatory scales, but also blood, serum, breath, urine, and feces probably carry infection during the entire course of the disease.

Now, as every case of scarlatina, even the mildest, may communicate a dangerous form of the disease, it is always wisest that every case should be treated as if it might develop a most dangerous epidemic. Six weeks of quarantine are none too long for an average case of scarlatina, and this should be indefinitely extended as long as desquamation may require. Seven years' experience in one of the orphan asylums of Chicago has convinced the writer that this is not only theoretically possible, but actually does prevent the spread of the disease, for never during these years has there been a general epidemic of scarlatina in the asylum, although sporadic cases have been not infrequent. In such institutions isolation can be more effectually carried out than in private families, but the effort should be made, and is usually attended with the happiest results. Long ago Dr. Budd wrote in reference to scarlatina: "Time after time have I treated this fever in houses crowded from attic to basement with children, who have nevertheless escaped infection by the simple method of isolation." Reliable statistics show that 50 per cent. of the children thus protected escape infection, and still better results ought to be obtained by local and personal disinfection added to isolation.

Disinfection of the sick-room should never be omitted. For this purpose J. Lewis Smith highly recommends volatilization of the following mixture in boiling water:

R̄. Acidi carbolicī
 Ol. eucalypti āā f 3j.
 Ol. terebinthinae f 3vj.—M.

Sig. A tablespoonful to be added from time to time to a pan of hot water, to be kept boiling on a gas stove or grate fire.

The sick-room should be the largest, most sunshiny, best-ventilated room in the house, and, if possible, should have an open fireplace. All curtains, pictures, ornaments, and furniture not absolutely necessary for the comfort of the patient should be removed before the child is placed there, and no one but the nurse and physician allowed to enter. The nurse should wear a loose wrapper and cap, to be dropped inside the door should she be compelled to meet other persons for any purpose outside the door.

An ordinary bed-sheet, tacked by one edge over the door and kept moistened with a 2 per cent. solution of carbolic acid, has apparently been helpful in preventing the spread of the disease in asylum practice, where, the writer agrees with J. Lewis Smith, the "area of contagiousness is small, and hence the disease is more easily quarantined than either measles or pertussis."

For disinfection of the patient J. Lewis Smith recommends as a local disinfectant to the faucial mucous membrane corrosive sublimate, 2 grs. to a pint of water (1 drachm containing $\frac{1}{84}$ of a grain). This may be used as a gargle, or as a spray from a hard-rubber atomizer. The same solution may be employed for cleansing the nasal cavities. The writer's preference for faucial application is a solution of eucalyptol in peroxide of hydrogen (gtt. xv to f3j), used in the cup of an ordinary steam atomizer. The same solution may be applied upon a swab to the fauces if there be extensive necrosis; or, diluted with an equal amount of water, it may be used for washing out the nares with a douche or fountain syringe.

Others speak highly of 50 per cent. boroglycerin for topical disinfection of the throat, and all sorts of more energetic disinfectants have been recommended (mineral acids, chlorine-water, galvano-cautery, etc.) with less obvious justification.

The frequent anointing of the body with some form of non-irritant antiseptic ointment in order that the action of the skin may be encouraged, restlessness allayed, and the scattering of the scales reduced to a minimum, is strongly advised. Such an ointment as carbolic acid, grs. 20, thymol grs. 10, to vaseline and lanoline each half an ounce, may be favorably employed. This should be applied at least twice daily, the skin having been previously cleansed with warm water in which a little soda is dissolved. J. Lewis Smith speaks highly of the following:

R_y. Acid. carbolicæ
 Olei eucalypti āā ʒj.
 Olei olivæ ʒvij.—M.

Sig. For inunction every three hours.

Even the old-fashioned fresh lard or ham-rind will be found grateful to the patient and helpful to the health officers. An excellent and more elegant prescription is:

R_y. Thymol gr. x.
 Ol. theobromæ ʒj.
 Alcohol q. s.—M.
 Ft solutio.

Sig. For inunction twice or three times a day.

Disinfection of the room in which the patient has been is scarcely less important than that of the patient, since the virus of scarlet fever is so tenacious in its potency that it will persist for years in houses or rooms not properly disinfected. If the walls are papered, they may be rubbed, as is done by paper-cleaners, with slices of rye bread, which will remove microbic spores and scales; or, better, if possible, they should be repapered, calcimined, or whitewashed. Previous to this, sulphur—1 lb. to each 100 cubic feet of room-space—should be burned in the infected apartment, which should be kept closed for eighteen hours thereafter.

The efficiency of sulphur dioxide as a disinfectant is greatly increased by

combining with it the vapor of water in a hermetically closed room (Squibb). Hence the room should be closed as tightly as possible by pasting strips of paper over the door-jambs and keyholes before burning the sulphur candles. To increase the efficiency of the sulphur dioxide by its union with aqueous vapor, the candles may be placed on bricks in an ordinary wash-tub partially filled with water, and allowed to burn in the closed room until they go out for want of oxygen. After the room has been opened and aired as fully as possible, it ought never to be reoccupied until the walls have been cleaned as previously directed or thoroughly scrubbed.

All sheets, bedding, towels, and articles that can be washed should be immediately thrown into boiling water after being used, and those articles that cannot be washed or boiled should be fumigated with sulphur, baked, or, still better, destroyed by burning, as should all toys and books used during the convalescence of the patient.

RUBELLA.

By WILLIAM T. PLANT, M. D.,

SYRACUSE.

PERHAPS there is no other disease of brief duration and benign character that has been so much written about and so variously named as rubella. It was for so long held to be related to measles or scarlet fever, or both, that the following names have naturally come from such views of its nature: French and German measles or scarlet fever; false, bastard, and hybrid measles; and epidemic roseola. These and others not worth remembering have come down to us. The German name, R \ddot{o} theln, is not, and will scarcely become, popular in America, because of its foreign appearance and difficult pronunciation. More attractive and satisfactory than all other names, and now quite generally adopted by English-speaking people, is that of *rubella*—a diminutive of *rubeola*, first suggested by Veale not many years ago. Indeed, the disease seems to have been waiting for a name, and only lately to have found a fitting one.

Previous to the middle of the last century rubella had had no very clear description or decided differentiation from measles, and almost down to the present time very many in the profession have regarded it as a sort of modified or mongrel measles. Now, however, through a happy agreement of medical opinion, the following points may be regarded as settled: 1st. Rubella, though much resembling measles and somewhat resembling scarlet fever, is a distinct entity, independent of these as of other diseases. 2d. It confers no protection against measles or scarlet fever, nor can either of these affections influence or prevent an attack of rubella.

Rubella is an acute, contagious, eruptive febrile disorder, due to a specific, but as yet unisolated, poison. It runs a rapid course and terminates almost always in recovery. It occurs, with few exceptions, but once in a lifetime; and commonly travels in epidemics of rather limited extent, though sometimes it spreads over large tracts of country in a short time; and not infrequently the observant physician encounters sporadic cases whose origin he cannot make out. At times it appears to part with its tendency to spread, though probably at all times its contagious property is less pronounced than that of measles.

Incubation.—The period of incubation varies greatly. Griffith observed a large institution-epidemic, originating from a child in whom the eruption appeared upon the day of admission. The first case was observed after five days, and 28 cases developed within eleven days after the earliest possible exposure. Other observers give periods varying from ten days to three weeks, the majority stating it to be from two to three weeks. The variability of this period, as Griffith has pointed out, offers a striking contrast to the fixed period of incubation of measles. Ordinarily, there are no symptoms observable during this stage. Occasionally, Squire states, the throat is complained of, and epistaxis and enlargement of the post-cervical glands may be observed.

Symptoms.—The prodromal stage is short, not more than a few hours, or a day at the most, though in many cases the eruption, like that of varicella, may be the first evidence of disease, especially in older children. When symptoms are observed they may comprise malaise, nervous irritability, slight suffusion of the conjunctivæ, perhaps with lachrymation and slight coryza, pains in the limbs, drowsiness, hoarseness, slight cough, sore throat, enlargement of post-cervical and post-auricular glands, with possibly an elevation of temperature of 1° to 3° F. Any or all of these symptoms may be wanting, and the first evidences of disease, as already stated, may be discovered in the rash.

The eruption of rubella appears first behind the ears and upon the forehead and face, especially upon the oral circle, spreading rapidly over the rest of the body, and reaching the legs last. When first discovered it may have already extended to the chest or abdomen. In rare cases the distribution of the rash may remain limited, as in a case observed by Griffith, in which, though the symptoms were severe, the rash could be found only upon the face and neck.

In appearance the rash is maculo-papular, pin-head to split-pea in size and pale rose in color. The spots are usually discrete, and are separated by areas of healthy skin; but in certain localities subjected to warmth and pressure they may become confluent and simulate closely the rash of scarlatina. Upon the chest and back the rash is usually darker red in color, and more profuse. From this, the typical appearance of the eruption, various departures occur, so that in one case the eruption of measles may be closely simulated, and in another the rash of scarlatina. This variability of the eruption is one of the most characteristic features of the disease. A study of these manifestations seems to warrant the recognition of two distinct types of variation from the normally developed rash: 1. *Rubella Morbilliforme*.—The eruption is discrete, the papules are nearly the size of a split pea, and more or less grouped, strongly resembling measles. 2. *Rubella Scarlatiniforme*.—Here the whole body is rapidly covered with a diffuse rash of bright rosy-red hue, which is raised somewhat from the surface of the skin, and often occurs in patches with well-defined margins. A few papules may often be found near the margins or within the reddened areas, and can be best seen perhaps on the fingers or wrists, or on the forehead.

In some cases, indeed, coalescence of papules may take place after some hours, and, as Tonge-Smith has pointed out, the rash may thus become blurred into a confluent blush on the second day, so as to be indistinguishable from scarlatina except from the history. Instances, however, will occur where the greatest minuteness of examination will fail to give conclusive evidence of the nature of the rash, particularly in the scarlatiniform variety.

In the development of the eruption variations will also be observed. Thomas states that ordinarily the maximum of the development of the eruption on different parts occurs at different times, following the sequence of its first appearance, and this opinion is shared by Hardaway, Emminghaus, Roth, and Griffith; other writers state that the eruption reaches its height on the second, rarely, as Cheadle asserts, on the third, day. The average duration of the eruption is fixed by Griffith at three to four days, though it often lasts a much shorter time, or may continue longer. As the eruption fades, slight brownish or yellowish pigmentations may be visible for a few days. Desquamation does not occur in all cases: according to the testimony of a few competent observers it has never been observed. It does, however, occur, but is always slight and furfuraceous in character, and is usually completed in a few days.

With the appearance of the rash or slightly preceding it other symptoms

appear. Catarrhal symptoms, referable to the conjunctivæ and nasal passages, are frequently present, but in slighter degree than is usual in measles, photophobia and marked coryza being quite rare. A loose cough is not unusual, but is distinctly less severe than that of measles. Sore throat is one of the most constant of the symptoms. It appears usually as a redness of the mucous membrane, especially marked about the uvula and upper portions of the anterior pillars; the tonsils are at times involved, and may be considerably swollen, giving rise to pain in swallowing. Griffith mentions an occasional eruption of small yellowish-red or brownish-red spots of pinhead size visible over the soft palate and uvula and the inner surface of the cheeks. This sore throat, however, is of little importance, and rapidly subsides, often to recur in the last stage of the disease. This secondary angina, according to Eustace Smith, is very characteristic of rubella. The tongue is either clean or has a thin yellowish-white coating, quite different from the characteristic "strawberry" tongue of scarlatina, the appearance of which is never simulated, according to the testimony of the great majority of writers. The temperature varies greatly, ranging up to 103° or 104° F., though, as a rule, rarely reaching 101° F., and often is not materially elevated. It is apt to be highest on the first or second day of the rash, and may then subside suddenly or fall gradually with the disappearance of the eruption. Pulse and respiration are rarely disturbed except in proportion to the rise of temperature.

Probably the most characteristic symptom of the disease is enlargement of the lymphatic glands, which to greater or less degree is present in almost every case. Those mainly affected are the post-cervical and post-auricular glands, but in many cases the axillary and inguinal glands are also involved. The swelling is hard, tender, and reaches the size of a pea. It is an early symptom, noticeable often on the first day, at times before the appearance of the rash, and practically it is never delayed beyond the second day. Griffith, however, believes that this glandular swelling, while a very constant symptom of rubella, is probably nearly equally as frequent in measles, and that it is by no means of as great diagnostic importance as is usually supposed.

Nausea and vomiting are extremely rare, and, though reported in isolated cases, should not be classed as symptomatic of this disease. The bowels also show no special disturbance of function. Slight œdema of the face may be observed when the rash is well marked. Itching of the skin is rarely present and is never troublesome.

Reinfection, or relapse, is of very rare occurrence, but has been occasionally observed within one to three weeks after the onset of the original attack.

Complications and Sequelæ.—In the disease as we know it at the present day complications or sequelæ directly traceable to it are extremely rare. Those most commonly mentioned involve the respiratory organs and air-passages, such as bronchitis and pneumonia, naso-pharyngeal catarrh, stomatitis, and permanent enlargement of the tonsils. Transient albuminuria is mentioned by Emminghaus, Kingsley, Reed, Cheadle, and others, while Mettenheimer, Tonge-Smith, and Squire doubt its occurrence, and Hardaway considers it entirely anomalous, if not due to mistaken diagnosis. Otorrhœa, ciliary blepharitis, and phlyctenular keratitis have been observed.

Prognosis.—Rubella is not a dangerous disease, and recovery is usually complete in a fortnight. Death occasionally occurs in severe cases and in some epidemics, and this from some serious complication.

Diagnosis.—From measles rubella may usually be distinguished by the short duration of its prodromal symptoms and the absence of marked catarrhal

symptoms and hoarse ringing cough; by the slight degree and the variability of fever; by the presence of sore throat and of enlargement of the post-cervical and post-auricular glands; and, in less certain degree, by the appearance of the eruption. From scarlatina it may be distinguished by the absence of vomiting at the onset, by the suffusion and faint congestion of the conjunctivæ; by the swelling of the lymph-glands, which occurs early, bears no relation to the severity of the faucial inflammation, and affects the post-cervical and post-auricular glands rather than those of the throat; by the appearance of the tongue, and lack of acceleration of pulse out of proportion to the elevation of temperature; by the absence of albuminuria; by the branny character of its desquamation; and, finally, by the appearance of the rash, which is more rosy in color and somewhat raised from the surface, often occurs in patches with well-defined margins, and is less burning to the touch.

Anomalous cases, however, arise which tax to the utmost the physician's skill in diagnosis, and the occurrence of other more typical cases in the same family may be the only means of distinguishing rubella from one or other of the more serious affections which it simulates.

Treatment.—Probably no disease needs less medical treatment. Its own direction being toward recovery, it may generally be safely left to follow it. The patient should be sent to bed, as well for the safety of others as for his own. As there is conjunctival irritation in most cases, the room should be darkened.

The diet should be light and bland, as toast, bread and warm milk, and various broths. Cool water should not be denied. If itching be troublesome, it may be allayed by frequent tepid bathing. Treat headaches by applying cloths wrung from cold lotions or by hot foot-baths made more effective by mustard.

The sore throat is well treated by the steam atomizer or by gargles, as follows:

R. Potassii chloratis ʒiiss;
 Glycerini fʒiij;
 Tinct. ferri chlorid. fʒss;
 Aquæ q. s. ad fʒviiij.—M.

Sig. Gargle once in three or four hours.

In a disease of such mild character it is doubtful whether any quarantine precautions need be advised, except to prevent loss of time and inconvenience in the school-room, where the disease is readily disseminated, often by cases passing without recognition. From this point of view two weeks after the beginning of the attack may be considered an ample period of quarantine.

CHICKEN-POX.

By WILLIAM T. PLANT, M. D.,

SYRACUSE.

VARICELLA, or chicken-pox, the lightest of the exanthemata and usually a disease of trivial importance, was first described as a distinct affection a few years before the close of the seventeenth century. There can be no doubt that it had existed from a period far remote, but it was not until then differentiated from small-pox and other eruptive disorders. Dr. William Heberden, an English physician who lived between 1710 and 1801, was the first to give a full and accurate description of this disease, though several writers before his day had described it less perfectly, and one of them, Dr. Richard Morton, gave it its earliest and best name of chicken-pox.

It is an acute, infectious, and transient affection, runs a definite course, and, with very few exceptions, occurs but once in the same person. Though it bears some resemblance to the lighter forms of variola, it has no relation to this disease, as has been abundantly proven by the observations of two centuries. Therefore, the name varicella, conferred upon it by Vogel in 1764, is founded upon error and is misleading.

It is essentially a pediatric disorder, as it only affects infants and young children—at least the writer does not remember to have met with it more than once or twice in adults. It may be regarded as quite a rare affection after fourteen or fifteen years of age. It travels in epidemics, often widespread, regardless of season, race, country, or climate, and of everything but age.

Incubation.—The incubative period is rather long. Henoch fixes its duration at 12 to 13 days; Gerhardt, 14 to 15; Eichhorst, 13 to 16; Strümpell, 13 to 17; and Semtschenko, 3 to 26. In cases of the inoculated disease d'Heilly has observed as short an incubation as 3 days; but with the affection as ordinarily contracted this period of latency may be assigned between the lowest and highest figures given by the authorities quoted, averaging 13 to 17 days.

Symptoms.—At the close of the incubation the active period of the disease is often ushered in with a little chilliness, aching of head and limbs, diminution of appetite or complete anorexia, and perhaps nausea. With these symptoms there is usually moderate fever—from 99° to 102° . It often happens, however, that the eruption is the first symptom noticed, no complaint of illness having been previously made by the child. Only in rare instances are the phenomena of invasion alarming or even severe. Decided chills, fever of high grade, and even delirium, are occasionally met with at the onset, and in one case under the writer's care the disease was ushered in by two severe convulsions. Some authors allude to this very rare mode of beginning. But, whether these first symptoms of invasion are usually mild or entirely unnoticed or exceptionally severe, they are of short duration, and the eruptive stage is soon established. As it first appears, irregularly scattered over the body, the

eruption consists of some small rose-red papules which very quickly develop into vesicles. This change is effected so quickly that very often the papular stage is over and the vesicular stage well under way before the eruption is discovered. The vesicles are seldom either numerous or large. Varying in number from a dozen or two to a hundred or more, they are scattered rather irregularly over the trunk, limbs, and scalp, but are most abundant upon the back. They seldom make very much show on the face. Frequently a few are found on the forehead and temples when all other parts of the face are quite free. Often, if searched for, some blebs may be found upon the mucous membrane of the mouth and fauces, where they quickly rupture and leave small ulcers. In the severer cases mild sore throat, laryngeal irritation, or slight hoarseness is sometimes noticeable, and in the light of the interesting observations of Boucheron and of Marfan and Hallé, to be presently referred to under the heading of Complications, it seems quite certain that hyperæmia of the upper air-passages and vocal cords may be present, and that vesicles may occasionally form upon the vocal cords, and possibly still lower down in the bronchial tree.

The vesicles of chicken-pox are quite variable in size: some are not larger than pin-heads, while others reach the size of small peas. It was presumably the resemblance in average size to the "chick-pea," or "cicer," of Southern Europe that suggested to Dr. Morton the name of *chicken-pox*.

The tegumentary covering of the vesicle is very thin, being composed only of the outer layers of the skin. It contains an alkaline serum of crystal transparency, whence another admirable name for the affection, "*crystalli*," and the German "*Wasserpocken*." It was long ago aptly said that the rash of chicken-pox suggests an appearance as if scalding water had been flirited over the surface, each drop having raised a small transparent blister. Some of the vesicles are surrounded by a narrow, often linear, and very pink areola; others rise abruptly from a surface of natural color.

A peculiar and distinguishing feature of chicken-pox is that the eruption comes out in successive crops. Before, or as soon as, the first vesicles have arrived at their full size others are just beginning; and this may be repeated twice or thrice, or even four times.

In the disease as ordinarily observed the vesicles never become pustular like those of small-pox, unless from scratching or other irritation, with consequent secondary infection; and, according to the usual teaching, they are neither partitioned nor umbilicated, as are those of variola, and are rarely so numerous as to become confluent. Walsh, however, quite recently has stated that the eruption may be macular and papular, with an inflammatory areola about the vesicles, which may be confluent, umbilicated, partitioned, and pustular, and finally may leave depressed cicatrices not unlike "pockmarks." In these times of general vaccination, with its protecting or, at least, mitigating influences, cases manifesting such peculiarities of the eruption must be regarded with grave suspicion, and the possibility of a masked variola must be taken into serious consideration, especially if the patient be an adult.

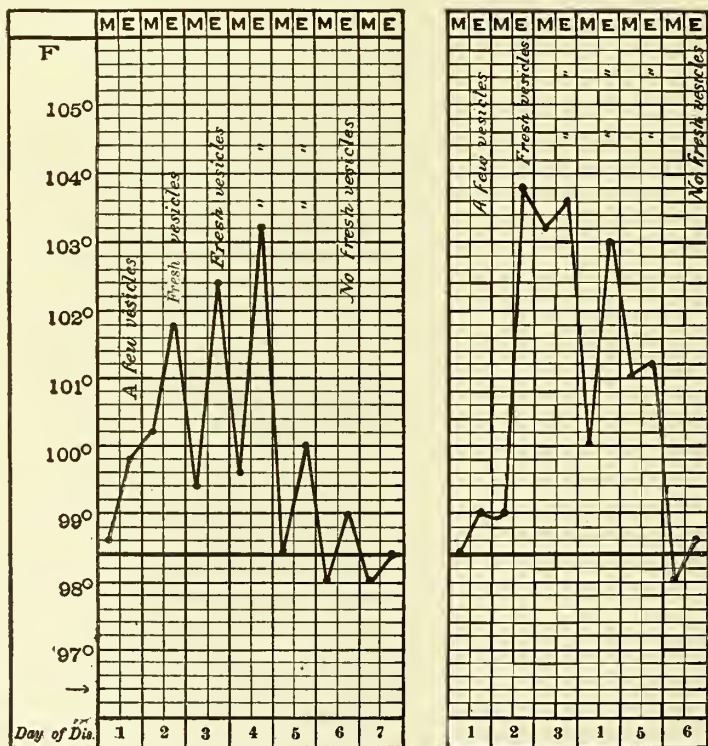
Another peculiarity of this disease is that, if the eruption is at all copious, many, perhaps most, of the vesicles abort and shrivel away before making much progress toward a completed development. I have observed that late vesicles are especially prone to abort. The other vesicles advance rapidly to maturity and enter on a speedy decline. The fluid becomes opalescent and turbid, and dries down into a thin yellowish crust that soon crumbles and falls off, leaving a temporary redness of the skin. In case of injury or irritation of a vesicle sufficient to cause a slight superficial destruction of the derm, and sometimes

even without this in vesicles of unusual size, healing is followed by a slightly excavated depression in the surface of the skin. Many persons bear in after-life one or more of these pits upon the face as a reminder of this childish malady.

During the eruptive stage the fever, which is almost uniformly intermittent in type, varies in degree with the acuteness of the attack and the extent of the eruption, mild cases, with only a few vesicles, being almost apyretic; severe cases, with a profuse eruption, being attended by a temperature of 104° or more. The usual range and duration of elevated temperature is illustrated in the accompanying charts (Fig. 1).

The whole course of chicken-pox seldom exceeds eight or nine days, or possibly ten or twelve at the most, and in the uncomplicated cases convalescence

FIG. 1.



Temperature Charts of Varicella (Ashby and Wright).

is rapid. As seen in young infants, however, especially in those already weakened by chronic digestive ailments, the disease, however mild in its manifestations, may be followed by a period of innutrition of more or less gravity. In hospital epidemics varicella is certain to add to the mortality among this class of patients.

Recurrence.—Second attacks of varicella are rare. In twelve epidemics studied by Semtschenko in Kasan, embracing 872 cases, only 14 instances of recurrence were found, the intervals varying from eight to eighteen months after the primary attack. In 5 other cases there were two subsequent attacks of the disease.

Complications and Sequelæ.—While ordinarily chicken-pox runs an

uneventful course in a previously healthy child, and is followed by rapid and complete recovery, recent observations have emphasized the fact that the kidneys may early present inflammatory changes, which may occasionally lead to a fatal termination. Attention was first drawn to this in 1884 by Henoch, who reported 4 cases of post-varicellous nephritis, one of which terminated fatally; and since then more than 30 cases have been published, principally by German observers. Cassel, one of the most recent of these, saw 6 cases out of 12 in a single epidemic in Berlin, in 1894, which showed albuminuria or actual nephritis, the earliest on the fourth or fifth day of the disease. Three of these were fatal—one, ten months old, from nephritis alone on the twelfth day, the others in association with pneumonia. Two other cases dying from nephritis have been recorded—one each by Högyes and Hagenbach, the latter referring to the condition as one of acute parenchymatous nephritis, while the former stated that the convoluted tubules and loops of Henle were alone affected. This testimony is sufficient to indicate the necessity for keeping close watch upon the urine during, and for a time after, the disease.

Von Starck has seen in a boy of two years, on the tenth day following the onset, a generalized oedema without albuminuria or other signs of nephritis. It was attributed to a peculiar action of the virus of the disease upon the vessels of the subcutaneous connective tissue, comparable to the condition signalized by Quincke and others as occurring after scarlatina.

The occurrence of a scarlatiniform erythema during the decline of the eruption has been occasionally observed. In one case lately reported by Comby albuminuria of four days' duration and suppuration in a submaxillary lymph-gland followed the erythema. The precise nature of this rare complication is still unsettled.

Suffocative laryngitis has been observed in 2 cases by Marfan and Hallé, as previously noted—one preceding, the other accompanying, the appearance of the eruption. The first case, a child of three years, was seen first after an illness of three days. The voice was hoarse and respiration difficult, with supra- and infrasternal recession. The throat was reddened, but otherwise not abnormal. On the fourth day the eruption appeared upon the surface, but the laryngeal symptoms increased, and necessitated tracheotomy. The child recovered. The second case, in a weakly infant of nine months, showed a similar affection of the larynx coincident with a profuse confluent eruption. Death occurred on the seventh day from acute diarrhoea and broncho-pneumonia. The autopsy showed a small round, deep ulcer at the posterior part of the margin of the right vocal cord. Boucheron also saw a case which proved fatal from spasm of the glottis, due probably to hyperæmia of the vocal cords.

Various other affections have been noted as occasional complications of this disease, among which may be mentioned furunculosis, osteitis, synovitis, otitis media, and submaxillary and cervical adenopathy, at times associated with inguinal bubo, and rarely going on to suppuration.

Varicella may complicate or be complicated by other infectious diseases: such combinations as varicella and pertussis, varicella and measles, varicella and scarlatina, varicella, measles, and pertussis, and varicella, measles, and diphtheria, are occasionally observed. Profuseness of the eruption alone may constitute a serious complication, as is illustrated by a fatal case in an infant of eight and a half months seen by Nisbet, who attributed its death to the fact that the eruption covered every portion of the body, producing the effect of an extensive burn.

Secondary infections are not very unusual. Of these erysipelas is the most common, and is always a grave complication. In a circumscribed epidemic

of 15 cases Bolognini observed 12 in which secondary infection of the vesicles by staphylococci and streptococci took place during the stage of desiccation, causing the vesicles to enlarge to the size of bullæ, which, breaking, gave issue to a thick creamy pus. In one case, the only one resulting fatally from abscess of the kidney, pure cultures of the streptococcus were obtained. All of these children had transient albuminuria, without other signs of nephritis.

Varicella Gangrænosa.—Among the secondary infections should be considered the rare condition which is described under this name. It was first brought to notice by Hutchinson in 1882, and was for a time thought to be peculiar to varicella; but subsequent observations have shown that an identical process may occur in connection with vaccinia, pemphigus, and other discrete pustular lesions. Dermatologists now describe the general affection under the name of *dermatitis gangrænosa infantum*. Tuberculosis, rickets, and inherited syphilis seem to exercise a predisposing influence, but it has been occasionally observed in apparently healthy children.

As seen in connection with varicella, it may begin while the vesicles are still present; it is then observed first upon the head or upper portions of the body. It will be noticed that ulceration has begun beneath the crust, and often a pustular margin with an inflammatory areola is found, resembling closely a vaccinal pustule. The destructive process extends in depth and periphery until it forms a black slough reaching an inch or more in diameter. After a time separation of the slough occurs, leaving a sharply-cut oval or roundish excavated ulcer. When the vesicles have been closely aggregated several gangrenous areas may coalesce to form larger ulcers of irregular contour.

When the gangrenous process begins as late as two weeks or more after the onset of the disease, after the varicellous lesions have healed, the ulcerations are more apt to begin upon the lower portion of the body, especially upon the buttocks and thighs. Pinhead-sized maculo-pustules first appear, which increase in size, rupture, and form crusts, under which the gangrenous process begins as in the case of pre-existing varicellous lesions.

In the severer cases, which begin early in the course of the exanthem, hæmorrhage into the vesicle precedes the other changes; and, with this, hæmorrhages from the nose, mouth, or stomach, as well as beneath unaffected portions of the skin, may be observed. Such cases run a rapid course, and terminate with symptoms of general pyæmia.

Of the pathology of gangrenous varicella nothing definite is known. There can be little doubt, however, that it results from a secondary infection, in the milder cases probably with the ordinary pyogenic organisms; and in the more malignant cases, such as those recently reported by Lockwood and Silver (*Archives of Pediatrics*, Sept., 1897), the coincidence of an acute blood-infection may be reasonably presumed.

Even in its mildest manifestations gangrenous varicella is a serious affection, but in the virulent types associated with marked blood-dyscrasia the prognosis is wellnigh hopeless.

Diagnosis.—It is usually only to settle this important question that the physician is summoned. Apart from variola or its milder manifestation, varioloid, eruptive vaccinia and herpes zoster are the only diseases with which varicella might reasonably be confounded.

From eruptive vaccinia, apart from the history of a recent vaccination, varicella may be distinguished by its successive crops of rapidly developed vesicles, which will have almost disappeared before the vaccinal lesions could have reached the height of their development and shown a marked areola.

From herpes zoster, its more general distribution, which does not follow the course of certain nerves, and the absence of pre-eruptive pain, should serve to differentiate it.

From well-marked variola and varioloid, varicella should be readily distinguished by a consideration of the following points of difference:

<i>Chicken-pox.</i>	<i>Variola.</i>
Only infants and young children affected.	All ages affected.
Invasion short; general symptoms usually very light.	Invasion three days; general symptoms severe.
Febrile stage transient, commonly highest at beginning of the eruption.	Initial fever falls with appearance of eruption, to be followed by the secondary rise with pustulation.
Eruption vesicular almost from the first.	Eruption papular for 3 or 4 days.
Eruption superficial: never shotty.	Eruption deep-seated: hard, shotty.
Seldom umbilicated.	Generally umbilicated.
Vesicles not distinctly multilocular.	Vesicles always multilocular.
Vesicles always discrete.	Eruption often confluent.
Eruption little on face, hands, and feet.	Eruption most on face, hands, and feet.
No pustular stage.	Pustular stage never absent.
Uninfluenced by vaccination or previous small-pox.	Prevented by vaccination or previous small-pox.

Mild and abortive cases of varioloid occur, however, and present the greatest difficulty in diagnosis. The invasion may be short, and so mild as to attract no attention; the lesions may be few and scattered; fever may be insignificant; and the vesicles may abort before reaching the pustular stage. In such a case error in favor of the milder disease is easily made, and may be followed by most disastrous consequences. Only a most careful study of the history and course of development of the attack can lead to a satisfactory decision; and if the patient should happen to be an adult, this fact should weigh decidedly in favor of the more serious disease.

Prognosis.—As a rule, when occurring in a previously healthy child, chicken-pox rarely gives rise to anxiety as to its outcome. Among debilitated, strumous, and syphilitic infants prognosis should be more guarded, lest the gangrenous complication supervene, the prognosis of which has been already stated.

Treatment.—A disease whose course and duration are fixed, and whose ending is almost always favorable, requires little aid from medicine. The child should be confined to bed during the active stage of the disease, and if fever be high a foot-bath should be given at the start, followed by a simple diaphoretic febrifuge. Except in the case of very young children, whose digestion is liable to passing disturbance from the disease, no special restriction in diet need be made unless the fever remains high for several days. As a rule, the eruption causes little irritation, and needs no treatment except a soothing dusting powder upon the back and upon the parts kept warm by the clothing. Upon the face large vesicles may be punctured early, and covered with a thin film of collodion to protect them against injury or secondary infection from scratching. For similar reasons the child's hands should be disinfected and the nails kept clean and well trimmed.

In all cases the urine should be watched, and from time to time during the course and convalescence should be examined for albumin or other evidence of nephritis. If convalescence be protracted and the child exhibit evidences of anæmia or disturbed nutrition, iron and cod-liver oil, with a bitter tonic, should be prescribed, with perhaps a change of air, preferably a short sojourn at the seashore.

Gangrenous varicella demands a much more rigid treatment. Constitutionally, the strength must be kept up by nourishing diet and by liberal stimulation, according to the indications, with some suitable preparation of alcohol, with strychnine, and with quinine. Locally, the gangrenous lesions must be treated with antiseptic and deodorizing washes, such as solutions of permanganate of potassium, peroxide of hydrogen, or bichloride of mercury, and kept covered with a protective ointment containing iodoform, ichthyol, or some other drug of this class.

Quarantine.—With a disease ordinarily so benign little effort is usually made to carry out quarantine. In many children's hospitals epidemics of varicella run their course unchecked, usually for want of sufficient facilities for isolation; and ordinarily the disease seems to have little disturbing effect upon the children except in the rare instances where a gangrenous complication occurs or among the athreptic babies, as already pointed out. In family practice a period of three weeks from the beginning of the disease may be considered a sufficient time for isolation. As with other infectious diseases, a thorough cleansing of the body and scalp and a change of clothing should be ordered before the child is allowed to mix with his playmates again. Without such precaution the danger of infecting others may last for some time, as was instanced in a case coming under the author's observation where the disease was communicated to an infant by a child who had recovered from an attack fully four weeks before the only occasion of their meeting and playing together.

VARIOLA AND VARIOLOID.

By C. G. JENNINGS, M. D.,

DETROIT.

VARIOLA, or small-pox, is an acute, specific, highly infectious disease, characterized by a typical range of temperature and a specific inflammation of the skin appearing usually on the third day of the disease as a papular eruption, which quickly becomes vesicular and finally pustular. The pustules desiccate, and leave permanent cicatrices wherever suppuration has invaded the deep tissue of the skin.

Etiology.—The nature of the contagium of variola is unknown; analogy, however, points to a micro-organism as the infectious principle. There is no evidence of the development of the disease *de novo*, each case being transmitted from a parent case in another individual. Individuals of both sexes and of all ages, unprotected by vaccination, are subject to the disease. Even the fœtus in utero does not enjoy immunity.

The disease is transmitted by direct contact, through the medium of infected articles and through the air. While scarlatina, measles, and other exanthemata will infect at the distance of only a few feet, small-pox has a striking distance that is very much greater. In the Sheffield epidemic (1887) the influence of the Sheffield hospital could be traced over an area having a radius of four thousand feet.

One attack, as a rule, renders an individual immune. In countries where the disease is prevalent a second attack is not uncommon. The writer saw a negro woman, ill with discrete variola, who was sadly disfigured by two previous attacks. The disease prevails most extensively among unvaccinated communities. The negro race is particularly susceptible. The disease is most infective during the periods of suppuration and desiccation. Although apparently independent of climate, small-pox is a disease of the winter and spring.

Pathological Anatomy.—The characteristic anatomical lesion of variola is found in the skin and mucous membranes. Small areas of congestion appear in the skin. The vessels of the corium dilate and become tortuous, and the connective tissue in the centre of the congested areas is thickened by œdema. Coagulation necrosis of the epithelial cells quickly follows, with thickening of the epidermis. These changes form the papules. Serum is poured out between the necrotic cells, and a vesicle forms. The changed cells form a meshwork in which the fluid is enclosed. Trabeculæ bind down the centre of the vesicle, while its periphery continues to distend, producing umbilication. Pus-cells form rapidly in the vesicle, and in a few hours it is transformed into a pustule. Inflammatory injection and thickening of the connective tissue surrounding the pustule now take place. If the necrotic process is confined to the superficial layers of the skin, resolution takes place without pitting. If the deep tissue is involved, a cicatrix results. Desiccation of the pustule follows, leaving a crust of dried cell-débris and pus adhering to the skin. Then the epidermis re-

forms under the crusts, the inflammatory injection and infiltration subside, the crusts drop off, and resolution is complete.

The process in the mucous membrane is the same. Perfect pustules, however, are rarely seen, because the macerated roof yields early to the pressure, and an aphthous-looking ulcer results, often covered by a pseudo-membrane. In hæmorrhagic small-pox the pustules contain blood, and extravasations may occur in the skin and mucous membranes at any point, and in the substance of all the organs. More or less intense congestion and septic inflammation may be found in the brain, liver, lungs, kidneys, and spleen.

Incubation.—The duration of the period of incubation of variola is, on the average, twelve days. Exceptionally it may be shortened to ten or lengthened to fifteen days. When transmitted by inoculation the disease appears on the eighth day or sooner. During the period of incubation the child, as a rule, shows no symptoms.

Symptoms.—The clinical history of small-pox may be divided into four stages: Invasion; eruption; secondary fever; desiccation or decline.

The stage of invasion is ushered in abruptly. Older children complain first of chilliness, and often there is a distinct rigor. The phenomena of severe fever quickly follow. In addition to the usual symptoms of fever there are headache of unusual severity, persistent vomiting, great prostration, and severe backache. In younger children and infants the disease begins with fever, great nervous irritability, and vomiting. Very often convulsions mark the onset of the disease. They may be frequently repeated, with intervals of stupor or delirium. The skin is dry or perspiring; the tongue coated, with dark-red edges. The bowels may be constipated, but often a sharp diarrhoea is present during the whole of the invasion stage. Abdominal pain and tenderness are frequent. Respiration is rapid. The pulse is full and quick, ranging from 120 to 160. The temperature quickly reaches a high point, ranging from 102° to 105° F., or higher. The high temperature is maintained during the invasion stage with but slight remissions. The maximum temperature of this stage is usually reached just before the appearance of the eruption. Partial paraplegia, numbness, and incontinence of urine and feces, are sometimes seen in children.

In children more frequently than in adults *initial* or *accidental* rashes appear about the second day, and cause much difficulty in diagnosis. The initial rash may be erythematous, simulating scarlatina or erysipelas; or macular, simulating measles. It is very evanescent, and usually ushers in an attack of varioloid. A number of observers have noted that the areas of skin affected by the prodromal rash escape the variolous eruption. Petechiæ from one-twelfth to one-fourth of an inch in diameter are sometimes seen in this stage of the disease scattered over the lateral thoracic and lower abdominal regions. This rash is often of grave prognostic significance.

The average duration of the stage of invasion is three days. In grave cases it is often shortened to two, while in varioloid it is often prolonged to four days. As a rule, the longer the incubation stage the milder will be the subsequent course of the disease. Notable exceptions to this rule are the delayed rashes of cases complicated by severe internal diseases, and, as Moore observes, of cases showing an early hæmorrhagic tendency.

The Stage of Eruption.—On the third day of the disease, with the variations noted above, the true rash of small-pox begins. The eruption shows first on the face, quickly extending to the scalp and neck. Exceptionally it covers the wrists early. After the face and neck, it next invades the trunk, extremities, and finally the palmar and plantar surfaces, taking from twenty-four to

forty-eight hours to cover the cutaneous surface. Rarely, in very young infants, the rash appears first about the lower part of the abdomen and on the inside of the thighs. Other exceptions to the usual sequence are sometimes met. The rash is most abundant on the face and on the back of the hands. It shows early and abundantly on irritated areas of skin.

The eruption begins as small, slightly raised, pale-red macules, and passes through four stages of development—viz. *macules*, *papules*, *vesicles*, and *pustules*. The macules in a few hours become fine, conical papules, pin-head in size and larger. The papular stage continues for two days. The well-developed papules are hard and shotty to the sense of touch, “feeling like grains of shot underneath the skin.” Gradual augmentation in the size of the papules takes place. On the third day a minute vesicle appears at the apex of the older papules; it rapidly grows, and transforms the papule into an umbilicated vesicle with cloudy contents. By the fifth day of the rash the fluid in the vesicles becomes turbid, and by the sixth day it is distinctly purulent. The eruption has now reached the pustule stage, or *stage of maturation*. The mature pock is globular and about the size of a pea. The increase of the contents has distended the chamber and removed the umbilication. The pustule is, in fact, a small abscess. It is usually surrounded by a swollen, red, inflammatory zone, the *halo* of the pustule.

Synchronous with the development of the cutaneous eruption a true variculous exanthem appears upon the mucous membranes. The visible mucous membranes are nearly always affected, and, in severe cases, the rash extends throughout the whole alimentary and respiratory tracts. The urethra, vagina, and conjunctivæ are often invaded.

With the appearance of the eruption a remarkable amelioration in all the symptoms takes place. The temperature rapidly falls, often reaching the normal point or a little above on the fifth or sixth day. This fall of the temperature is pathognomonic of the disease. The pulse loses its rapidity and the gastric and intestinal irritability subsides. In cases of severity the remission is less marked, and the severe symptoms of the incubation stage persist with but little relief. In discrete small-pox convalescence often sets in after three or four days of the mild febrile movement which follows the sharp decline of the beginning of the eruptive period.

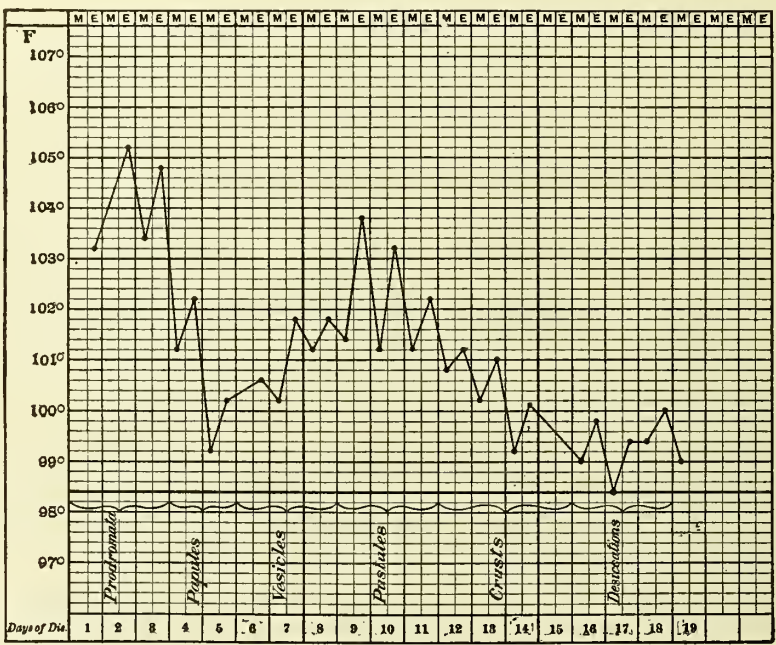
In children, with the beginning of the vesicular stage the eruption in the mouth and throat becomes a source of distress and danger. The vesicles rupture, and a streptococcus pseudo-membrane covers the resulting erosions and often extends over a large area of mucous membrane. Nasal and pharyngeal obstruction results, with distressing symptoms, and if the larynx be invaded, croup with dangerous stenosis may supervene.

In typical variola the maturation of the rash is accompanied by the onset of the *secondary fever* or *fever of suppuration*, which is of indefinite duration and varies in intensity with the severity of the attack. The child becomes restless and there is mild or active delirium. The temperature ranges from 101° to 104° F., with morning remissions and evening exacerbations. The pulse is quick and hard. Often the symptoms assume the typhoid type, with low delirium or stupor, a rapid, feeble pulse, and subsultus tendinum. A temperature that frequently rises above 104° during the stage of suppuration is of grave significance. (See Fig. 1).

The *stage of desiccation* or *decline* begins on the twelfth or thirteenth day of the disease. The pustules begin to dry up, the inflammation and swelling of the skin subside, the temperature gradually falls, and there is a general improvement in all the symptoms. Many of the pustules rupture and the

exuded contents form discrete or coalesced crusts. Cicatrization goes on underneath the crusts, and they finally drop off, leaving dark, violaceous blotches that

FIG. 1.



Temperature Chart of Variola of Moderate Severity.

are gradually changed to white, irregular, depressed cicatrices. The whole course of the disease occupies from three to five weeks.

Based upon the distribution and amount of the rash, variola is classified into—

(1) *Discrete variola*, in which the rash is scanty and the individual lesions are more or less separated from one another by healthy skin. The disease is rarely dangerous to life, its symptoms are mild, and its course is often interrupted before the development of the pustular stage. The secondary fever is absent or of short duration.

(2) *Confluent variola*, which is marked by an eruption that covers almost the entire cutaneous surface and invades the mucous membranes with great severity. The pustules upon the hands and face “run together, so that the epidermis is raised by a milky, sero-purulent secretion;” on other parts of the body the eruption is more or less discrete. The invasion stage is severe, and the rash appears as early as the second day. Severe vomiting and diarrhoea, stomatitis, salivation, pseudo-diphtheria, great and painful swelling of the face, hands, and feet, pyæmic abscesses, high fever, violent delirium, and great prostration are marked features of this type of the disease. The mortality is great, and convalescence is very slow and often interrupted by serious sequelæ.

In addition to these chief varieties we recognize—

(3) *Hæmorrhagic variola*, a malignant form of the disease, characterized by profound alterations of the blood, leading to the formation of petechial blotches and ecchymoses and more or less profuse hæmorrhages from the mucous membranes.

(4) *Varioloid* is variola modified in its course, duration, or intensity by vaccination, previous attacks of variola, or inherited insusceptibility. The invasion stage of varioloid is more irregular in duration than that of unmodified variola, and the symptoms may be so mild as to escape observation, or so intense as to simulate the onset of grave variola. Three types of variation in the clinical history of varioloid may be distinguished: (a) After an invasion stage of the severity of typical variola a copious eruption appears. With the appearance of the rash, however, a rapid defervescence begins, and the eruption is aborted in the papular or the vesicular stage. If it go on to the pustular stage, the pustules quickly run their course without causing much discomfort to the patient, and leave only faint cicatrices or none at all. Or, (b) the disease runs a course typical in all respects, but the pustules are few in number and the accompanying symptoms very mild. Again, (c) the symptoms of invasion are well marked. A trifling eruption of maculo-papules appears and quickly fades. Instead of rapidly convalescing, however, the patient shows a period of anæmia and mental and physical prostration out of all proportion to the preceding symptoms.

Complications and Sequelæ.—The complications of variola are few in number. Streptococcus invasion of the subcutaneous connective tissue may give rise to multiple abscesses, phlegmonous erysipelas, boils, and, rarely, in scrofulous children, to gangrene; the deeper structures, the joints, and the viscera may also be invaded. In children the most frequent complications are inflammations of the mucous membranes. Pseudo-diphtheria of the pharynx, nose, and larynx is frequent in severe variola; rarely the membrane invades the bronchi. Bronchitis and broncho-pneumonia, pleuritis with resulting empyema, purulent otitis media, and pericarditis or endocarditis often occur. Conjunctivitis is present in all bad cases; sometimes the inflammation is very severe, and results in ulceration of the cornea and destruction of the eye. Enterocolitis is often the cause of death in infants.

Diagnosis.—Typical variola in the eruptive stage presents no difficulty of diagnosis. Mild and atypical cases, however, are often very perplexing. The invasion stage may be mistaken for a continued fever or pneumonia. The sharp pain in the back, the vomiting, and the marked nervous symptoms should put the physician on his guard. The initial erythematous rash, coming on the second day, and the vomiting, are very like scarlatina. The small, often irregular, and very rapid pulse, the peculiar tongue, and the pharyngitis are distinctive of scarlatina. The rash of scarlatina, again, has a different initial distribution; it first appears on the face, neck, and front of the chest.

An initial macular rash, or the papular stage of variola, may simulate measles. In measles the gradual onset of the invasion stage, the tendency to sleep, the catarrh of the conjunctival and respiratory mucous membranes, the absence of the backache, severe headache, and vomiting, are distinguishing features. With the appearance of the rash in measles the fever and all the other symptoms *increase*; in variola they *decrease*. The "grisolle sign" is a certain means of distinguishing the papules of variola from the macules of measles: "If upon stretching an affected portion of the skin the papule becomes unpalpable to the touch, the eruption is caused by measles; if, on the contrary, the papule is felt when the skin is drawn out, the eruption is the result of small-pox."

The differential diagnosis of variola and varicella sometimes presents great difficulty. Varicella is characterized by a short period of invasion, the eruption usually being the first indication of ill-health that the child manifests. The varicellous vesicle is located beneath the most superficial layers of the epi-

dermis. The macular stage of varicella is short, and the macule is soft and but slightly elevated above the surface. The vesicle does not become pustular, but remains filled with clear or opalescent fluid for twenty-four or forty-eight hours, and then dries into a light, easily-detached crust. The distribution of the vesicles, abundantly over the back and sparsely on the face and hands, is very characteristic of varicella. Occasionally only the greatest care will enable the physician to differentiate between these two diseases. No one symptom or manifestation can be relied upon, but all the points in the history and development of a given case must be carefully considered.

Prognosis.—The frequency of complications involving the mucous membranes in children, and their feeble powers of resistance make the prognosis of variola in early life very grave. According to Moore, the disease is most fatal in unvaccinated children under five years of age. The younger the child the graver the prognosis. "The influence of vaccination for good is unquestionable, the mortality being 50 per cent. among the unvaccinated in general, 20 per cent. among the badly vaccinated, and only $2\frac{3}{10}$ per cent. among the efficiently vaccinated" (Moore). Hæmorrhagic and confluent variola are very fatal. The complications that unfavorably influence the result are—pneumonia, empyema, multiple abscesses, septicæmia, pseudo-membranous laryngitis, and entero-colitis. Favorable cases present a mild or no secondary fever, and are not prolonged by complications.

Treatment.—There is no drug that will prevent the development of variola in an infected individual. The efficacy of vaccination in arresting or modifying the disease after exposure is a disputed question. Curschmann has no confidence in the measure. Welch, however, from an experience in 159 cases, believes it to be of great utility, and his results warrant the use of the measure in every person exposed to variola: "In order that protection shall be complete it is necessary that the insertion of the vaccine lymph should be made almost immediately after the reception of the contagion; but if made at a somewhat later date a modifying effect may be obtained. No part of the incubation period should be considered too late to make use of this remedy, since this period is sometimes prolonged beyond its usual limit, in which case a late vaccination may prove of value" (Welch).

A child ill with small-pox should be placed in a very well-ventilated room of a temperature of 65° to 70° F. The strictest attention should be paid through the whole course of the disease to the smallest details of the hygiene of the patient and the sick-room. If the attack be severe, the hair should be closely cut. The diet should be light and nutritious. Effervescent waters, milk and seltzer, sour wine, champagne or lemon-juice and apollinaris, Belfast ginger-ale, and egg-water form agreeable and nutritious drinks. During the period of invasion the febrile symptoms, vomiting, headache and backache, and the nervous phenomena may demand treatment. A gentle cathartic should be given at the onset of the disease. A febrifuge, like tincture of aconite, spirits of nitrous ether, or a solution of acetate of ammonium may be given in proper doses. Gastric irritability may be controlled by effervescing citrate of potassium, chloroform-water, or subnitrate of bismuth. Chloroform-water and morphine are very useful, combined as follows:

R_y. Morphinæ sulphatis gr. $\frac{1}{8}$.

Aq. chloroformi f ʒij.—M.

Sig. A teaspoonful may be given every half hour to a child of five years.

Insomnia or convulsions demand the administration of chloral or bromide

of potassium. Baths, temperature 95° F., are most useful to control the fever and nervous symptoms, and they may be repeated every four, six, or eight hours as may be necessary. One of the coal-tar antipyretics may be given. They have a remarkable power to control the pain, nervous symptoms, and fever at the onset of an acute disease. Given in proper doses and in selected cases, their effect is only for good.- Applications that irritate and redden the skin are to be avoided. An ice-bag or a cold-water coil to the head lowers temperature and relieves cerebral symptoms.

During the eruptive stage, after the development of the secondary fever, the same conditions for internal treatment are met. The fever is to be controlled, preferably by the bath, made lukewarm or cool as the season and the condition of the patient dictate. Cool sponging, cool compresses, or the wet-sheet may replace the tub. The coal-tar antipyretics are to be given with caution. Delirium and convulsions are to be met by bromide of potassium, chloral, or the bath; insomnia, by these remedies or sulphonal. When there is intestinal irritability, chloranodyne is an admirable sedative. Quinine and the tincture of chloride of iron in full doses have the confidence of able practitioners as being useful to combat septic symptoms. Variola with mild secondary fever will not usually demand alcoholic stimulants. In grave cases moderate stimulation should be begun early, and as the strength wanes under the influence of continued septic absorption the alcohol should be pushed to the full limit. A child of five years will take from two to four ounces of whiskey or its equivalent in the twenty-four hours, sometimes more.

The nose, naso-pharynx, and throat should receive strict attention to relieve inflammation and avoid septic absorption. Irrigation of the pharynx with solution of potassium chlorate, boric acid, or witch-hazel should be begun early. The writer finds a solution of listerine and hydrogen peroxide one of the most satisfactory local remedies for pseudo-membranous and septic conditions of the mouth and throat, for example:

Solution of hydrogen peroxide(15 vol.),	
Listerine	each 1 part.
Water	6 parts.

This solution should be thrown into the pharynx with an all-soft rubber syringe, until thoroughly cleansed, every one, two, or three hours. This is the most satisfactory way to cleanse a child's throat. The same solution, with double the quantity of water, may be used in the nose with the same syringe. When such thorough cleansing is not demanded, the spray from an atomizer will serve, but it should not be trusted in severe cases.

To limit the development of the pustules and to prevent septic absorption and pitting a great number of methods of local treatment have been proposed. Secondary streptococcus-infection of the pustules without doubt plays an important part in the cutaneous destruction, septic absorption, and deep pus-formations; careful cutaneous disinfection during the papular and vesicular stages of the eruption will tend to limit this secondary infection. The skin should be bathed twice a day with soap and water, and this followed by sponging with a boric-acid solution 1:20, diluted listerine, or corrosive sublimate 1:2000. Omitting the soap, the baths, varied to suit the condition, may be continued during the whole course of the disease. Carbolic acid is an excellent antiseptic and cutaneous analgesic. It is one of the most efficient remedies for the relief of the itching and burning that accompany the development of the rash. Compresses of antiseptic gauze, wet with a hot or cold

solution, 1 : 500, may be kept constantly applied to the skin. Carbolic acid may also be used in solution with glycerin or in an ointment. An ointment of 4 parts of salicylate of sodium and 100 parts of cold cream is commended. Antiseptics may also be used as a spray or in the form of a powder, as subnitrate of bismuth, boric acid, or a compound of aristol 20 parts, talc 100 parts. Powders are most useful in the late stages of the eruption. Early opening of the pustules is a measure advocated by many writers. It seems rational thus to treat the pustules as small abscesses—to open them early, at least upon the hands and face, and treat them antiseptically. A wet compress of antiseptic gauze applied after evacuation and thorough cleansing with a three-volume solution of hydrogen peroxide would certainly prevent additional destruction of the corium from pus-microbe invasion.

In the stage of decline iron, quinine, and strychnine, highly nutritious food, and moderate stimulation are demanded. Convalescence is often slow and interrupted by complications. Arsenic, cod-liver oil, malt, iron, liquors, and supporting treatment generally are necessary. The various complications and sequels should receive the most approved medical and surgical treatment.

Quarantine.—A child with small-pox should be immediately isolated, and a rigid quarantine maintained until the skin is free from crusts and complicating suppurations have healed—a period of from five to six weeks. Confinement in a contagious diseases hospital gives most certain protection to a community, although perfect isolation can be maintained in a private house. For this purpose the highest, best-aired, and most remote room should be selected, opening indirectly, if possible, to the rest of the house. Sheets wet with an antiseptic solution should be kept hung over the doorway. All direct communication of the nurse and patient with other members of the family should be interdicted. Clothing, dishes, excreta, etc. should be disinfected before being taken from the room. All members of the infected household should cease direct communication with the outside world, and all exposed individuals should be quarantined for a period of fourteen days after exposure.

VACCINIA; VACCINATION.

By THOMPSON S. WESTCOTT, M. D.,

PHILADELPHIA.

VACCINIA, or cow-pox, is a contagious eruptive disease of the cow, characterized by a more or less profuse eruption, upon the udder and teats, of papules which develop into vesicles, and these, by drying, into crusts, or, through rupture, into open ulcers. By inoculation of lymph from its vesicle the disease is communicable to man, and is capable of conferring upon him immunity from small-pox more or less complete and lasting.

History.—In the closing years of the eighteenth century, among all the civilized nations of Europe and their colonies, the practice of inoculating for small-pox had become the accepted therapeutic procedure for modifying the ravages of this then most familiar and loathsome of diseases. The operation was not, however, always successful in producing mild cases of the disease, and even in its most favorable manifestation the communicated affection was still variola, capable of being transmitted to others by effluvium, and necessitating careful isolation, nursing, and medical treatment. So common was small-pox that, according to the philosophy of the times, every individual had either passed through, or was destined some time to experience, an attack of the disease. In 1776, Edward Jenner, an English country practitioner living at Berkeley in Gloucestershire, was first attracted by a popular belief, common among the dairy-hands of this county, that any one who had contracted cow-pox from milking cows affected with this disease was insusceptible to small-pox, and was not a successful subject for variolous inoculation. This tradition seems to have been quite well known among the dairy-hands of Gloucestershire and the neighboring counties, and to have been noted by other practitioners throughout the farming country. Intentional inoculation of cow-pox had even been performed before Jenner's attention was directed to the matter: Robert Fooks, a butcher of Bridport, as related by Pearson, had submitted to the inoculation by means of a charged needle, as early as 1771, and Benjamin Jesty, a farmer of Yetminster in Dorset, in 1774 inoculated his wife and two sons with the cow-pox as a preventive of small-pox. But it was not until the subject received the careful study and experimentation of Jenner, culminating in his celebrated *Inquiry*, published in 1798, that the practice of inoculating cow-pox was established upon a clinical and what, at least for the times, must be called a scientific basis. The story of Jenner's struggles to convince his contemporaries of the value of his observations forms a most interesting and instructive chapter in the history of medical progress. The discovery spread with wonderful rapidity throughout the civilized world, and it stands to-day as one of the greatest blessings that human thought and observation have conferred upon mankind.

Etiology.—"Spontaneous" cow-pox, the term ordinarily though not very accurately applied to cases of vaccinia occurring naturally in the cow, is an

occasional disease among dairy herds. It is spread by contact, being usually carried from one animal to another by the hands of the milkers, who in this way are themselves liable to accidental inoculation. For this reason the affection is almost exclusively confined to milch-cows, and the eruption limited to the udder or teats, although young calves or adult bulls may be readily inoculated upon the belly, and exhibit phenomena differing in no way from those observed in the cow.

The exact nature of vaccinal disease is a question which has been the subject of repeated theorizing and experimentation since the time of Jenner, and even at the present day no consensus of opinion has been reached. Jenner held that cow-pox was occasioned by the accidental conveyance of the virus of "grease," an eruptive disease of the heels of the horse, to which also he attributed, on conjectural grounds merely, the origin of human small-pox. According to his view, a vaccinated person was a small-poxed person who, instead of suffering from the humanized and virulent form of the disease, had contracted it in its primitive mild character. This theory, at least in regard to its ingenious attempt at the etiological unification of cow-pox and small-pox, can be dismissed as a curiosity of medical history.

A second theory considers vaccinia as a distinct disease of the cow originating in a specific contagium, and being in no way related to or capable of being originated by any other contagium, however closely its phenomena may be simulated. It is evident that its rejection or its acceptance is to be based upon the proof or refutation of other theories, and thus it can be more readily discussed side by side with the third and remaining theory.

This theory, which offers in many respects the most rational view of the question, regards cow-pox as small-pox modified and attenuated by passing through the system of the cow. There can be no doubt that variola can be artificially communicated to the cow, and can give rise to a vesicular eruption resembling in all physical respects the lesions of spontaneous cow-pox, and that virus from these vesicles can be conveyed to man, and produce at the points of inoculation local effects in all appearance identical with those produced by cultivated vaccine-lymph. Experiments of this kind are now quite numerous recorded, among which may be mentioned the successful variolations of the cow performed by Gassner in 1801, and after him those of Thiele of Kasan, Ceely of Aylesbury, Badcock of Brighton, Martin of Attleboro, Mass., Voit, Reiter, and many others. In some cases the virus thus obtained, when used for experimental inoculation upon human subjects, especially in the early removes, showed undoubted evidence of being variolous by giving origin through infection to fresh cases of small-pox some of which were fatal. Martin's variola-lymph produced quite an epidemic of small-pox in Attleboro, Massachusetts, in 1836, and Reiter's experiments in Munich in 1839 had a similar sequel. It is certain, however, that if in the selection of a variolous virus the same care be exercised as was habitual with experienced small-pox inoculators like Sutton and Dimsdale, a variolation of the cow may be effected which will give origin to a lymph that need not necessarily convey infection to those not inoculated. This was shown in the experience with Badcock's variola-lymph; and, as Crookshank remarks, identical results were obtained by Adams in many cases where lymph from a mild or "pearl" case of small-pox was taken as a primary virus for successive arm-to-arm inoculations, without having been first passed through the cow.

This whole subject was carefully investigated in 1865 by the Lyons Commission under the direction of Chauveau, who, even in 1891, still showed himself the most distinguished champion of the dual nature of the two diseases.

The result of the investigation of this committee unequivocally pronounced upon the autonomy of cow-pox and the impossibility of converting small-pox into cow-pox. A more recent investigation of the question by Fleming, a well-known English veterinarian, confirmed the conclusions of the Lyons Commission. The question is not, however, by any means settled. Even as recently as 1892, Hime of England and Haccius and Éternod of Switzerland, published careful studies in support of the older view, and, excepting in France and America, the theory of the identity of the two diseases seems to be gaining ground.

To complete the subject it may be stated that several years ago Depaul of Paris established the fact that horse-pox, a febrile eruptive disease of the horse, was capable of being conveyed by inoculation to the cow, and giving rise to a lesion indistinguishable from that of cow-pox. Constantin Paul, indeed, for a time used such virus for vaccination, but the practice fell into disuse after the discovery of a case of spontaneous vaccinia at Beaugency.

Pathological Anatomy.—The structure of the vaccine pock resembles that of variola (Cornil and Ranvier). It is formed by the softening and liquefaction of the epidermic cells, which appears to be caused by the micro-organisms which early occupy the centre of the pustule. There is a central necrotic zone, a middle zone characterized by tumefaction of the cells, and a peripheral zone of irritation showing multiplication of nuclei (Pincus). The cavity of the pock is partitioned or multiloculated, and its base, thickened and infiltrated with lymph, constitutes the “vaccinal pulp.” The derm is always infiltrated with leucocytes. The lymph is a clear, transparent liquid up to the fifth day in the cow and till the seventh or eighth in man; it maintains its infective qualities at a low temperature, but loses them quickly in warmth. Histologically, it contains leucocytes, red globules (after the eighth day), granulations and cellular débris, free nuclei, and micro-organisms.

Keber in 1868, and subsequently Chauveau and Burdon-Sanderson, observed the existence in lymph of minute rounded organisms to which the terms vaccinads or microspheres have been applied. Keber attributed to them the specific properties of the lymph. More recently (1890) the experiments of Straus, Chambon, and Ménard have shown that lymph from which these bodies had been removed by filtration loses its infective power, even when injected in quantity beneath the skin, so that it may be concluded that these micro-organisms are the agents of infection. No distinct microbe, however, has as yet been satisfactorily isolated. In 1883, Quist cultivated upon alkaline serum a coccus, which, when inoculated upon a child, rendered it refractory to subsequent vaccination. Voigt (1885) isolated three micro-organisms, of which one, a coccus, was found capable of causing typical experimental cow-pox in the calf, from which the same organism was again obtained. Garré (1887) confirmed the results of Voigt, cultivating a coccus which existed in a pure state under the derm subjacent to the pustule, and which caused cow-pox in the calf, but not in man until after passage through the calf.

Varieties of Lymph.—Practically, there are two sources from which vaccine-lymph may be obtained—either directly from the bovine through the agency of vaccine farms especially established for its propagation, or indirectly therefrom after passage through the system of one or more human beings, the healthy infant being the medium usually chosen. Lymph from the so-called cases of spontaneous cow-pox is very rarely to be had, and is said to be untrustworthy in its infective powers; while variola-vaccine must still be considered as of experimental value merely, and not to be ordinarily employed. At the present day it may be said that in no essential respect is humanized virus to

be preferred to animal lymph, if we except its slightly greater promptness of action, which may, however, have some value in time of epidemics. The possibility of the transmission of syphilis through humanized lymph derived from a syphilitic patient, while exceedingly rare, is still a constant danger, and pleads strongly against the use of any humanized virus except from an unimpeachable source. In selecting lymph, either from the calf or from the human vaccinifer, a characteristic vesicle from the fifth to the seventh day should be chosen.

Symptoms.—When carefully selected and cultivated vaccine-lymph is introduced by inoculation into the human system, the following phenomena will be normally observed: At or close to the site of inoculation at the end of the second or beginning of the third day a slight papular elevation is observed; by the fifth or sixth day this has become a distinct vesicle, of bluish-white color, with rounded elevated edges and a cupped central depression—the so-called umbilication. By the eighth day the vesicle is perfected, and is then circular, pearly in color, and distended with a colorless lymph, the central depression remaining well marked. On or about this day appears the areola, a reddish blush of the skin surrounding the pock to a distance of several inches, and accompanied by induration and swelling of the underlying connective tissue. After the tenth day the areola begins to fade, the vesicular contents begin to dry in the centre, the process extending to the surrounding lymph, which becomes opaque and gradually desiccates, until by the fifteenth day a hard brownish thick scab is formed, which is gradually detached and falls in the fourth week. A circular, depressed, pitted, or sometimes radiated cicatrix remains. If there have been several points of inoculation close together, a compound vesicle of irregular shape may result. Even with a single surface of inoculation one or more additional vesicles may arise at some little distance from this point.

Constitutional symptoms are almost always notable to some degree in a case of primary vaccination. The temperature may rise one or two degrees on the third or fourth day, and remain elevated for several days. In children restlessness, irritability, and loss of appetite may frequently be noticed. The axillary glands or the inguinal glands, depending upon the choice of the arm or leg for operation, will usually show some swelling and tenderness for several days. In many cases, mostly those of secondary vaccination, the constitutional symptoms are more severe; the fever higher, with transient delirium; nausea or perhaps vomiting; and distressing headache. Itching of the skin round about the pock is commonly experienced, perhaps throughout the whole course of the case, and this may be so severe as to constitute a true pruritus.

Irregularities in the Course.—Various irregular manifestations of the pock have been described by earlier writers, but in later years, since the more general employment of animal lymph, these irregular forms have become much rarer. One peculiar abortive form, the raspberry excrescence, should be mentioned. Here the pock is rather slow in appearing, and never reaches full development, but becomes a flat, hard, reddish papule, resembling a *nævus*, and finally, after weeks or months, disappears without cicatrix. It is probably an abortive form, and does not protect against small-pox or subsequent vaccination.

Another irregularity is the so-called eruptive vaccinia, in which there is a generalized eruption of pocks, the disease manifesting itself as a true exanthem. Very rarely cases have been observed in which the susceptibility of the skin was so great that repeated accidental auto-inoculations took place from the merest scratches of the nails.

Complications.—*Inflammatory phenomena*, due to traumatism, irritation, infection, or special conditions of the system predisposing to cutaneous disease, are at times manifest. These may vary from a simple erythema to intense phlegmonous inflammation or ulceration and gangrene, with septic absorption. Injury to the pock before complete maturation may be followed by a gangrenous condition of the underlying derm, sometimes giving rise to a peculiar moat-like depression around a central elevated core. Mothers are very prone to attribute any irregularities or unusual violence in the maturation of the pock to “bad virus.” Occasionally, especially when human crusts have been used, this may be a just charge; but it can be authoritatively stated that complications arising from impurities of the lymph will almost invariably show their presence long before the pock has reached its full development, usually within a few days after the operation.

Erysipelas is very prone to infect vaccination wounds. It may appear as early as the second or third day, and in this case the prognosis is especially grave. Vaccination should never be performed when erysipelas is prevalent, except in face of the greater danger of variola.

Glandular Enlargement.—The natural involvement of the axillary and cervical glands, usually insignificant, may in certain subjects become extreme, and even go on to suppuration during maturation or toward the decline. In children of strumous habit vaccination may act as the exciting cause of chronic enlargement and cheesy degeneration of glands in these chains.

Abscess and boils may follow in various parts of the body, especially in children of tubercular tendency.

Eczema and other skin affections are apt to be aggravated or relighted by vaccination. Various roseolous rashes may be observed during the maturation of the pock, and are only important as requiring differential diagnosis from intercurrent and perhaps more serious affections, such as erysipelas, scarlatina, and rubella. Impetigo contagiosa has been observed not infrequently, and seems to bear some relation to vaccinia, which is as yet not clearly understood.

Syphilis.—Chiefly to Viennois in France and Hutchinson in England are we indebted for the demonstration that syphilis may be communicated by humanized virus through contamination with the patient's blood, which, as Ricord has shown, is always present in the lymph. Accidental conveyance of the disease by imperfectly cleansed instruments used for vaccinating is also to be mentioned.

The treatment of complications will not differ from that to be employed in the conditions occurring independently of the vaccinal disease.

Method of Operation.—Inoculation can be accomplished in numerous ways. Some practitioners advocate a series of superficial cross-bar incisions made with a sharply-pointed lancet or the back of the point of an ordinary bistoury; others employ a sharply-pointed rake-like instrument made for the purpose, while tattooing with a sharp needle point has been advocated. Altogether the most satisfactory method of preparing the spot for vaccination, and one which robs the little operation of its terror to children and mothers, consists in gently scraping away the external horny layer of epidermis with the edge of a bistoury or lancet held obliquely to the surface. For this purpose a dull instrument is sometimes advocated, but a sharp edge is more effectual and expeditious. An area as large as the little finger-nail can be readily abraded in this manner without giving rise to a whimper on the part of the child. The abraded surface should be slightly red and glazed by the outpouring of lymph, but no blood should be drawn. The next step is the inoculation of the lymph. In arm-to-arm vaccination the lymph is directly transferred from the pock to

the abraded surface. When the dried animal virus is used, it should be liquefied by dipping into cold sterilized water just before the surface is prepared, so that in the brief interval it may become completely softened.

Any portion of the cutaneous surface may be chosen for the insertion, but customarily the outer aspect of the left arm over the insertion of the deltoid is selected. For cosmetic reasons in girls the leg is often preferred, and in this case a point over the head of the fibula or over the junction of the two heads of the gastrocnemius is the usual choice. The primary vaccination of the infant may be undertaken at any time. In the face of an epidemic the new-born babe should be vaccinated within twenty-four or forty-eight hours after birth, and, as the experience of Wolff has shown, in such cases humanized lymph is to be preferred as producing less constitutional disturbance. Ordinarily, however, the operation may be deferred until about the third month when the child is in good physical condition and before the disturbances of dentition have commenced.

Protective Power of Vaccination.—The experience of the past one hundred years offers the most just and conclusive evidence of the power of vaccination as a preventive of small-pox. From one of the commonest and most virulent of diseases small-pox has become in civilized countries one of the rarest of the exanthemata. A most significant fact in favor of vaccination is given by Gay in a study of small-pox in London. He states that in the last forty years of this century, owing to improved sanitation, epidemics of measles, scarlatina, diphtheria, and whooping-cough have all undergone a decrease, but that this is only a small fraction of that which has occurred in small-pox, their highest figures not amounting to a tenth part of the decrease of small-pox—a result which is dependent upon only one possible cause, vaccination. Drysdale states that during the epidemic in Berlin in 1872 and 1873 the mortality rose to 243 and 263 per 100,000; then, vaccination in the first year of life and revaccination in the twelfth being made compulsory, during the first year of enforcement (1875) the mortality fell to 3.6 per 100,000, to 3.1 in 1876, and to 0.3 in 1877.

The protective power is not absolute in all individuals, nor can the period of protection be stated for any given case. Marson, whose experience with small-pox in London was very extensive, stated that the disease was more fatal among those whose scars were imperfect or few in number than in those showing well-marked and multiple cicatrices. While some doubt of the value of this theory may be expressed, it would seem wisest to vaccinate in all cases by at least two insertions, sufficiently far apart to prevent coalescence during development of the pocks. As a general rule, it may be stated that immunity in the great majority of cases will be attained by revaccination every four or five years, and always when small-pox becomes epidemic. If absolute immunity from small-pox be not conferred, the course of the disease will be greatly modified and ameliorated. In some very rare instances vaccination and revaccination seem to offer no obstacle to the development of severe variolous disease. According to Biedert, after a successful vaccination immunity is secured in about eight days. Vaccination after infection with variola does not guard against the development of the disease, but if done eight days before the eruption appears the evolution will take place benignly.

PAROTITIS.

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By the term "parotitis" is to be understood an inflammation of the parotid gland. By the inelegant term *mumps* we usually understand an acute infectious disease, often epidemic in character, in which the parotid gland is always inflamed, other glands being also involved occasionally. If it were possible to dislodge the term "mumps" from the mind of the profession and the public, it would be in the line of progress, for, like many other terms which cling to medical nomenclature, it is inaccurate, inelegant, and would be inexpressive were it not for its arbitrary association with acute epidemic parotitis.

This affection is usually regarded as one of the diseases of childhood. It is unfortunately true that many mothers think it necessary that their children must experience this and several other infectious diseases at some period of their childhood, forgetful of the fact that disease is always to be avoided if possible. It is true that one attack of epidemic parotitis usually furnishes immunity from others of the same character, but until we are further advanced than at present in the science of preventive inoculation it will not be wise to encourage the acquirement of infectious disease from such a motive. Small-pox, and possibly hydrophobia and tetanus, furnish exceptions to this rule, and the day is probably dawning when the list can be lengthened.

Epidemic parotitis is not limited to the period of childhood. Many epidemics are recorded in which it prevailed exclusively among men. This is especially true of soldiers in garrisons and barracks. Two such epidemics are recorded by Girard in which the testicular complication was severe, and others by Gnasco, Dogny, Jourdan and Laurens. Males suffer with it more frequently than females.

But parotitis is not necessarily an infectious disease, for there is a form which is purely traumatic and limited to the parotid gland, and another which may be called an irritative form, in which malignant disease in or near the gland incidentally causes true inflammatory action with infiltration and induration. Of this form nothing further need be said in this connection, the consideration of the subject being limited (1) to its traumatic, (2) to its infectious, aspect.

Pathological Anatomy.—Writers upon pædiatrics have remarked the incompleteness of the knowledge of the anatomy of this subject. This is due to the small number of fatal cases, excepting those in which the disease has occurred as a complication, and in which, from gangrene or abscess, the gland-structure is more or less completely destroyed. Virchow studied the disease in 1858, and his work is fundamental with reference to anatomical knowledge at that period. The development of bacteriological science has modified all our knowledge concerning infectious disease and its effects. In general it may be said, with Ziegler, that the anatomical appearances are those which are due to inflammatory, serous, and cellular infiltration of the inter-

alveolar fibrous tissue of the glands, issuing either in resolution, fibroid induration, suppuration, or gangrene. Bamberger describes the gland as enlarged, red, swollen with exudate in the interstitial tissue, the acini fused together, and the cellular tissue of the entire gland involved. In severe cases the entire glandular substance is involved and converted into a fleshy dry tumor. The exudate may be absorbed, the gland resuming its normal size and consistency, or the exudate in the cellular tissue may become thickened and organized, leading either to permanent increase in size or to atrophy.

Etiology.—The two varieties or forms of the disease to be considered are: (1) the traumatic, (2) the infectious.

(1) *The traumatic variety* is the result of blows or bruises, with more or less effusion of blood into the gland and surrounding tissues. The inflammation and swelling may be extensive, especially in syphilitic or strumous subjects, the great sensitiveness of the glandular system of such individuals rendering them peculiarly liable to disease of this character even when the injuries received have only been of moderate severity. It may also be the result of burns about the face and neck or of the application of irritating chemicals and caustics. This form of the disease is entirely distinct from the infectious, and illustrates the fact, which for some time was in dispute, that inflammatory conditions are quite possible without the influence of micro-organisms.

(2) *The infectious form* of the disease may be simple or immediate, symptomatic or metastatic. That parotitis may be a complication of so many other conditions is an argument against the proposition that it is always caused by a specific microbe. There is scarcely an infectious disease in which it may not so appear. It may complicate pneumonia, diphtheria, and typhoid fever, each of which has its specific cause; hence we are obliged to refer it to that very convenient class of diseases known as mixed infections, in which the limitations to one who is not a bacteriologist are as yet rather vague. It is quite proper to refer to the work which has been done with the view of placing its etiology upon a definite basis (*i. e.* from a bacteriological standpoint).

Pasteur found a bacterium in blood taken from patients with this disease, but inoculations of animals with cultures obtained from it were negative. Bordas described a bacillus found in the blood which he termed *bacillus parotidis*. In certain phases of its development it assumed an S or Y shape; when divided the ends became enlarged. It died at a temperature of 140° F., and its spores at 194° F. Its development was arrested in 1:500,000 solutions of mercuric bichloride. Cultures were made from the saliva of parotitic patients, and were rich in the microbe. The investigations of Capitan and Charrin in this field have been more extensive than others, and have to a great degree furnished a basis for other work. They first examined the blood, saliva, and urine from six cases. In the blood were found small, mobile microbes in great numbers, most of them being spherical, but some rod-shaped. Similar bodies were found in the saliva, while in the urine they detected neither albumin, sugar, nor microbes. In 1881, after a study of the blood in thirteen additional cases, they were able to confirm their previous discoveries. They particularly described a bacterium two to three thousandths of a millimetre long and also a small micrococcus, the microbes appearing singly, doubly, and in chains. Cultures of the microbes were successfully made, but inoculations of dogs, rabbits, and guinea-pigs were negative. These discoveries were verified by Védérès, Bouchard, Netter, and Boinet, the latter finding the microbes in the blood of fifteen patients, also in pus from an abscess of the nucha. Ollivier found the microbes in saliva, urine, and blood from three subjects, and suggested that failure in the inoculation of animals was due to the insusceptibility to parotitis of all species of animals

upon which experiments had thus far been conducted. He believed that we could now see in parotitis not the simple effect of cold, or a manifestation of the rheumatic diathesis, or a propagation of a phlegmasia of the mouth, but an infectious disease caused by a specific agent and propagated by the diffusion of that agent. Jaccoud has expressed himself almost equally hopefully.

In the simple or immediate form, which is the usual one in most epidemics, the contamination of the atmosphere with the infectious elements, especially in schools or barracks, in which the air-supply is deficient, explains its dissemination. This statement harmonizes with the fact that it is most prevalent in damp and cold weather when the windows and doors of houses are closed and the tendency or the necessity is to remain in-doors. The elements of the disease are also carried from house to house in the clothes of physicians and visiting friends. This explains the prevalence of epidemics in sparsely-settled localities. Infection is probably acquired in respiration, and those who are mouth-breathers are the more susceptible. Whether the long period of incubation which follows the reception of the infective influence means retention of the elements in the ducts of the salivary glands or in the glands themselves, or whether there is a process of germination within the blood and localization in the glands, we do not know. The latter is the more reasonable hypothesis from the analogy with other infective germs which are known to develop in the blood. As in all other infectious diseases, the intensity is governed partly by the activity of the infective elements and partly by the resistance of the individual.

In the secondary, metastatic, or symptomatic variety of infectious parotitis the inflammation is a complication of a pre-existing disorder. The list of diseases in which it may play such a rôle is a long one, including the infectious diseases in general, besides nephritis, pneumonia, meningitis, and surgical injuries of all kinds; for in all of them sepsis, and hence infection, are possibilities. As an evidence of extensive or general systemic infection it is a symptom of grave significance. With the diathetic diseases, tuberculosis, syphilis, and rheumatism, its significance is less grave than with the acute infectious diseases. In this variety we cannot refer to a specific microbe as its origin. Some of the conditions with which it may be associated have such origins (diphtheria, pneumonia), and whether the complicating parotitis is due to the irritating effect of such specific germs which have been retained within the gland, or whether it is caused by those germs (*streptococcus*, *staphylococcus*) which produce severe inflammation wherever localized, we do not as yet know.

Incubation.—The period of incubation of parotitis is a long one, but it varies with the resisting power of the individual and the virulence of the infective material. The long period of incubation, with the complicating conditions which may arise in the mean time, may delay the determination of the diagnosis. J. Lewis Smith regards the disease as primarily a systemic infectious one, with an incubation period of nine to twenty-one days; A. Jacobi fixes it at two to three weeks; Dauchez, at fifteen days; Roth, at eighteen days; and Nicholson reports a case in which an interval of six weeks elapsed between the involvement of the two parotid glands.

Symptoms.—The long period of incubation may be attended by symptoms of impending trouble. This is especially true with young children. There may be malaise with moderate rise of temperature for several days, and with very young children there may be convulsions, especially if digestive disorder coexist. With glandular swelling come also induration, sensitiveness, pain on motion of the neck or jaw, loss of appetite, restlessness, and insomnia. With the progress of the inflammation infiltration of the gland and the surrounding tissues increases, and fever is more pronounced. These symptoms

may continue for a week, and gradually subside, or the duration may be less prolonged. The induration will gradually disappear and normal conditions be resumed, or the gland may be permanently enlarged or it may atrophy. In a certain number of cases abscess or gangrene will ensue, the gland will be destroyed, and the final result be fatal; but in the great majority these are cases in which the system is so saturated with septic products that the outcome would be fatal even if parotitis did not exist.

The inflammatory action which involves the parotid glands may include also the other salivary glands, and even the cervical lymphatic glands. These complications are frequently overlooked, being overshadowed by the more extensive and apparent affection of the parotids. The appearance of an individual with parotitis is sufficiently characteristic: there is glandular swelling, with hardness and pain; the swelling may be considerable or inconsiderable, and of course the disfigurement of the face and neck will be governed accordingly. The pain is constant and severe, especially in young children; deglutition is difficult and often impossible on account of its painfulness. If abscess develops, the pain has the acute throbbing character of abscess-formation everywhere. Pain in the contiguous structures of the ear is almost always a marked feature of the disease, and the nearness of the carotid artery and cerebral meninges introduces elements of danger which must always be remembered, for serious results in this quarter are by no means unknown. Considering the possibilities of serious consequences, the small percentage of fatal cases when the disease is uncomplicated is quite remarkable.

Complications.—In the traumatic form, in which the inflammation is a simple one, complications are unusual. The inflammation subsides, as such conditions do elsewhere, the result being resolution in the mild cases and suppuration in the severe ones, especially if the tissues have been bruised and broken. In the epidemic infectious form complications are extremely common, the genital organs being most frequently implicated. Thus with males there is often an involvement of the testicles, spermatic cord, and inguinal glands; with females, the mammæ, ovaries, labia majora, and inguinal glands. These complications may not be evident until the symptoms in the parotid gland have begun to subside. In a recent epidemic in which one hundred and seventeen cases were observed by Demme, two were fatal from gangrene of the parotid glands; in three there was abscess of the cervical glands; in two there was acute nephritis. Musgrove and Slagle each saw a fatal case complicated with uræmia. P. Smith saw two cases which were followed by insanity, and Parrott one which was complicated with orchitis and meningitis. F. W. Brown records an epidemic of twenty cases in a boys' school, ten of which were complicated with orchitis. Jackson observed four cases complicated with influenza. This latter complication is more frequent than is generally supposed. The writer recently saw such a case in an infant fourteen months old.

Among the sequelæ of the disease Joffroy mentions peripheral neuritis, with paralysis of the extremities lasting four months. Rotch and Moure each saw two cases of deafness; and Dufour, inflammation of the lachrymal glands. The evidence is therefore abundant that we have in parotitis an infectious disease with multiple localization.

Treatment.—If the disease be, as it appears, an infectious one, we have, as yet, no method of treatment for aborting it. When the symptoms are apparent, the indication is to relieve them as they arise. The pain may be soothed by small doses of Dover's powder or paregoric, or phenacetin combined with salol. Hot applications to the inflamed parts are always grateful, and the surface may be kept moist with anodyne liniments. The bowels must be

kept open, fever may be reduced with aconite, and the diet must be fluid and concentrated. Hot liquids will usually be preferable to cold, and will be more quickly assimilated. The skin should be kept active by daily warm baths, by alcohol, and by gentle friction. The opiates suggested will usually be sufficient to relieve restlessness and induce sleep. As soon as the acute symptoms have subsided the nutrition should be improved as rapidly as possible, and a tonic of iron, quinine, strychnine, and arsenic will be indicated.

Quarantine.—An important practical question is that relating to the time in which patients with infectious parotitis should be isolated. This especially concerns children who are attending school. A recent paper by Rendu is devoted to this aspect of the subject. His studies have led him to believe that the time of greatest danger of contagion is at the close of the incubation period, at least twenty-four hours before the disease can be diagnosticated. Sevestre and Comby had reached this same conclusion. If this be a fact, Rendu's opinion that it is irrational to keep children out of school three weeks after the symptoms of the disease have subsided is a just one, and teaches that isolation should be limited to a period included between the time when the first symptoms appear and the time when the active symptoms have subsided.

WHOOPIING-COUGH.

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Synonyms.—Pertussis; Tussis convulsiva; Hooping cough; Chin cough.

Whooping-cough is a zymotic, contagious disease of childhood, characterized by a catarrh of the respiratory mucous membrane and a peculiar paroxysmal cough.

No description of any disease resembling pertussis can be found in the writings of the Greeks, Romans, or Arabians, and it seems probable that the failure to mention such a peculiarly characteristic disorder is proof that it did not then exist at all, or at least in parts of the world with which medical writers were acquainted. In fact, no account of it is found until Baillou, in 1578, described an epidemic which occurred at Paris, and spoke of it as an affection not previously known. Little or nothing more was heard of it for about a hundred years, when Willis wrote of "*tussis puerorum convulsiva*" in such a manner that its nature and its identity with the pertussis of the present day can admit of no doubt. Epidemics did not become frequent until the eighteenth century, but the disease then rapidly spread, and by the middle of that century had become widely diffused. From that period onward it has been steadily on the increase, until it constitutes at present one of the commonest diseases of childhood.

Etiology.—There are certain factors which seem to exercise a decidedly predisposing influence upon the development of pertussis. There is a very distinct tendency shown for it to occur in epidemics, which appear at intervals of about two years, yet with no great regularity in this respect. The disease may, however, occur sporadically, although such cases are always the result of some preceding case. In the larger cities it is practically endemic, although at times greatly more prevalent than at others.

The previous occurrence of the disease in an individual precludes the development of a second attack. Nevertheless, undoubted exceptions to this rule have been occasionally reported, though they are certainly rare.

Whooping-cough is more prevalent in the civilized portions of the world, but its absence from any region seems to depend rather on the fact that it has not yet been carried thither than on any conditions of climate or of race which are unfavorable to its existence. The influence of season has been much disputed, and the evidence is conflicting. It is certainly no powerfully predisposing factor. The station in life and the general hygienic conditions existing appear to be without influence, except in so far as the ill-ventilated houses of the poor may possibly favor the increase of the germs in number or in virulence, even as the crowding and lack of isolation certainly favor their diffusion.

The previous state of the health seems to possess some predisposing power.

Most observers agree that weakly, sickly children more readily contract whooping-cough than do those in good health. It is a well-recognized fact, also, that there is an intimate association between epidemics of measles and of whooping-cough, and it is very widely believed that the existence of the first disease strongly predisposes to the later development of the second. Whether or not the association is an accidental one is still unsettled. The actual presence of any other disease is certainly no bar to the occurrence of pertussis. As with other infectious disorders, there exists a certain individual susceptibility to it. Some children never contract it, though often exposed.

Age exercises a powerful influence on the development of whooping-cough. By far the greater number of cases occur before the sixth year. After this time the frequency of occurrence diminishes very rapidly, and after the tenth year it is comparatively infrequent. West estimates that over one-half the cases develop under the age of three years. It is sometimes seen in adults, but this is rather uncommon; the rarity being due partly to the fact that so many have suffered from it while children, and partly to a lessening of the susceptibility with advancing years. It is not common during the first six months of life. It is, however, distinctly more liable to occur at this time and up to the age of one year than are the other infectious disorders of childhood. There are even a few well-authenticated cases reported in which it appeared to have been contracted during foetal life.

It has been widely stated that girls are more liable to develop whooping-cough than are boys. Statistics, however, are somewhat at variance, but certainly show that there is no very material difference in the number of each sex attacked.

The sole exciting cause of pertussis is contagion, and so powerful is this contagiousness that by far the greater number of children exposed to the disease will contract it. It is contagious during any part of its course, but particularly in the paroxysmal stage. It is least so in the terminal stage. The nature of the infectious principle can best be discussed when considering the pathology of the affection.

As a rule, actual contact with, or close approach to, the sick child is necessary for its development in a second case, but even a momentary exposure of this sort is often sufficient to ensure an attack. Several observers have claimed that the disease does not spread readily in well-ventilated and roomy hospital wards. My own experience has not been at all in accord with this. The infectious germs appear to be located in the secretion of the respiratory tract, and are spread by this and by the expired air. Cases have been reported which show that whooping-cough is mediately contagious through a third party or through handkerchiefs or clothing which have presumably been infected by the sputum of a patient. It is probable, however, that the disease is rarely contracted in this way.

The contagiousness of pertussis extends slightly to the lower animals, and cases are on record in which these have contracted it from the human subject.

The path by which the germs enter the system is not certainly known. Although nearly all the evidence is in favor of the respiratory tract, the few published cases of pertussis in the new-born indicate the possibility of their entrance in other ways, as by the foetal circulation.

Pathology.—There are no post-mortem appearances characteristic of pertussis. The most constant change found is redness and swelling of the mucous membrane of the respiratory tract, with the presence of a considerable quantity of viscid mucus. There is often observed a tendency to congestion of various parts of the body, due to the disturbance of the circulation which naturally

attends the paroxysms. There are also found the various lesions corresponding to the complications which have existed during life.

The nature of pertussis has been a much-mooted question, and is not even yet entirely settled. It has been frequently claimed that the disease is a functional disturbance of either the pneumogastric, phrenic, recurrent laryngeal, or sympathetic nerves or of the medulla. According to this view, it is simply a neurosis. Other writers have viewed it as a simple bronchial catarrh due to cold merely, with which is associated a certain nervous element. Enlargement of the tracheal and bronchial glands has also been urged as the cause of the disease, through their irritating pressure upon the terminal filaments of the pneumogastric nerve.

The eminently contagious nature of whooping-cough, its occurrence in epidemics, the existence of a period of incubation, and the immunity from second attacks seem to prove beyond a doubt that it is to be classed among purely infectious disorders. Although this is the view which has recently found very general acceptance, it is by no means a new idea. Even Linnæus attributed pertussis to the presence in the nose of the larvæ of insects. Poulet discovered bacteria in the expired air of patients with pertussis. Letzerich found a micrococcus in the sputum which he believed to be the specific germ, and was able to produce the disease in animals by introducing the secretion into the trachea. Deichler claimed that there was always present in the sputum an organism of the nature of a protozoon which possessed amœboid motion. But, although other investigators have repeatedly described various organisms as existing on the respiratory mucous membrane, the researches of Afanassiew in 1887 have attracted the most attention. This observer isolated a short bacillus, which he named the *bacillus tussis convulsivæ*, and of which he was able to obtain pure cultures upon various media. Animals inoculated upon the respiratory mucous membrane with these cultures exhibited some of the symptoms of the disease and developed catarrhal conditions of the respiratory tract, with a tendency to broncho-pneumonia. These observations have been confirmed by others, and a toxine has also been reported as present in the urine of patients with pertussis which is identical with that produced by Afanassiew's bacillus.

Even though it be admitted as most probable that some micro-organism is the cause of the malady, it is by no means clear how the symptoms are produced or where the principal seat of the infection is. Some writers have claimed that the trigeminal nerve is in a sensitive state, and that it is the irritation of its terminal filaments by the infectious catarrhal process on the nasal mucous membrane which brings on the paroxysms by a reflex action. Others, again, have stated that the bronchial mucous membrane is the portion of the respiratory tract chiefly involved, and that the terminal filaments of the pneumogastric are those irritated. The careful investigations of Meyer-Hüni and of v. Herff, however, indicate that the catarrhal inflammation is most pronounced in the mucous membrane of the nose, larynx, and trachea down to the bifurcation, but especially so on the posterior wall of the larynx in the inter-arytenoid region, the so-called "cough region." In the production of the cough it would seem probable that a small quantity of mucus, perhaps arising from below, accumulates upon the surface of the "cough region," and there irritates powerfully the hyper-sensitive filaments of the superior laryngeal nerve. Through a reflex action a series of clonic spasms of the expiratory muscles is then set up. At last the crowing inspiration occurs, this depending upon a spasm of the glottis, which, in its turn, proceeds from an irritation of the convulsive centres in the medulla. This process is repeated again and again until the offending secretion is expelled.

The presence of this secretion does not seem, however, to be an essential to the production of the cough, since paroxysms may be brought on by excitement and other causes. This appears to indicate that the irritation of the superior laryngeal nerve may be central, due to systemic infection. A great preponderance of the nervous element of the disease over the catarrhal is further shown by the greater frequency with which the paroxysms occur at night, since this condition very possibly depends upon a less degree of resistance of the respiratory centre during the night, and a consequent greater ease with which convulsive expiratory efforts are brought about.

We therefore clearly have to do in whooping-cough with an infectious, catarrhal process which affects particularly, and produces an unusual sensitiveness in, the mucous membrane presided over by the superior laryngeal nerve. But still more prominent is a great excitability of the nerve itself and of the other nervous portion of the respiratory apparatus, this being probably due to the circulation in the blood of some noxious substance, the product of the infecting germs, which possesses a special power over the portion of the nervous system which controls cough. The apparent value in many cases of local treatment directed to the respiratory mucous membrane indicates that the abode of the germs is in this region, whence the poisonous products of their growth are absorbed. On the other hand, the existence of pertussis in the new-born, the result of foetal infection, points to the presence of the microbes themselves in the circulation and in other parts of the body besides the respiratory tract. From this point of view their situation in the latter region would be a localization entirely secondary to the general systemic infection and, so to speak, *excretory*. Which of these theories is correct cannot as yet be determined, although the resemblance of the disease to other infectious disorders certainly supports the latter view.

Incubation.—A period of incubation precedes the development of the symptoms. Its exact duration cannot be easily determined, since the onset of the disease is so insidious, and statements vary in regard to it. It is clearly somewhat variable in length, and probably lasts from two to seven days, with an average of three to four days.

Symptoms.—It is customary to divide the course of the disease into three stages: 1st, the catarrhal or premonitory stage; 2d, the paroxysmal or convulsive stage; and 3d, the terminal stage or stage of decline. This classification is convenient, but somewhat artificial, since the stages only very gradually pass into each other, and their duration cannot, therefore, be accurately determined.

1. Catarrhal Stage.—There is little in this which is characteristic of the disease. The child gradually begins to exhibit symptoms of a severe cold, with malaise, perhaps slight hoarseness, stoppage of the nose, tickling in the throat, sneezing, irritation of the eyes and a dry, annoying cough. Fever is generally slight and apt to come on in the evening only. Although it has been claimed that the elevation of temperature is an evidence of the infection, it is more likely that the degree of fever is dependent solely upon the intensity of the catarrh.

Under treatment there may be a temporary improvement in some of the symptoms, but all of them soon return in force, and the cough particularly is troublesome and gradually grows worse in spite of medicine given. As days pass by it shows a greater tendency to occur in long, severe paroxysms, and is also much more annoying by night. On examination of the chest only a very few râles may be heard. Nothing, indeed, is found to account for the severity of the cough. Sometimes, though less commonly, the first stage is characterized

by a severe bronchitis, with corresponding auscultatory signs and the presence of high fever.

The duration of the first stage averages about two weeks, but it is subject to great variations. Sometimes only two or three days elapse before the child begins to whoop. The younger the age, the shorter, often, is the duration of the catarrhal stage. In some instances the disease never passes beyond the first stage, the diagnosis in such cases depending largely upon the existence of the affection in other members of the family.

2. *Paroxysmal Stage*.—The complete development of the paroxysmal cough marks the beginning of the second stage. The exact time of onset is, as already stated, often difficult of determination. Except for the rarer cases in which the whoop never occurs, it is convenient and most customary to date the paroxysmal stage from the first appearance of this symptom.

The paroxysm of pertussis—or the “kink,” as it is frequently called—is very characteristic. Just before it begins the child seems anxious and irritable, or perhaps very quiet. It experiences some sort of a warning sensation, as a pain in the region of the sternum, or nausea, or a tickling in the nose, or a similar sensation in the larynx with an irresistible desire to cough. It at once drops its playthings, runs to its mother or nurse, or grasps some near object for support; or, if asleep, quickly rises, sits upright, and begins to cough. Sometimes, however, the cough seems to come suddenly, without the premonitory sensation. The cough consists of a number of short, explosive expiratory efforts very rapidly following one another, and without any inspiration between them. These continue so long and are so violent that the face becomes turgid and cyanotic, the tongue is protruded and driven against the teeth, saliva flows from the mouth, the eyeballs are prominent, the eyes water, and the pulse becomes rapid and small. The paroxysm lasts a few seconds until at last both cough and all respiration cease. Then comes a peculiar, loud, crowing inspiration, the *whoop*, which is the result of the air passing through the spasmodically closed glottis. Immediately there begins another series of expiratory efforts, to be again followed by the whooping inspiration; and this process repeats itself several times. The later series of expulsive efforts is accompanied by abundant expectoration of ropy mucus and very often by vomiting. As the paroxysm ceases the cyanosis disappears, and the child is often left pale and exhausted for a short time; but if it is strong and otherwise well it soon resumes its play. Sometimes a crowing inspiration immediately precedes the first series of expirations. Occasionally, too, after the attack seems to be over there is a period of rest for a moment, and the whole process is then repeated. A series of paroxysms may thus continue for as long as ten to thirty and even more minutes. The usual duration of an attack, however, is from a few seconds up to one or two minutes. The swelling of the face, the puffiness of the eyes, and some degree of blueness of the tongue persist more or less between the paroxysms, and may constitute quite notable features of the disease. In bad cases the paroxysms may be attended by hæmorrhage from the mouth or nose or beneath the conjunctiva or elsewhere. Involuntary voidance of urine or fæces may be occasioned by the violence of the attack.

The frequency of paroxysms and their intensity vary greatly. In mild cases there may not be more than six to twelve in the twenty-four hours, while in the severer ones they may number from forty to eighty. They are always more numerous at night. An attack of coughing is often brought on by exercise, crying, singing, loud speaking, eating or drinking, excitement of any kind, a sudden change of temperature in the air, or the breathing of air

overloaded with carbonic dioxide. Depression of the tongue with a spatula, producing gagging, is very apt to bring on an attack.

The general condition of the patient does not suffer materially in mild cases. Sometimes, however, there is much exhaustion from the frequent coughing and the loss of sleep, or vomiting may so regularly follow the paroxysms that the nutrition suffers greatly and emaciation becomes marked. In the milder cases vomiting does not at all interfere with the appetite, and the child is soon ready to eat again; so that quite sufficient food is retained for the bodily needs.

More or less fever may occasionally be present in the second stage, especially at night, but, as a rule, fever is absent, and if continuously present makes the existence of some complication probable. The urine in whooping-cough sometimes contains sugar and frequently albumin. It was at one time claimed that it was always saccharine. Auscultation of the chest in the interval between the paroxysms reveals nothing abnormal, or only the presence of a few mucous râles. During the whooping inspiration nothing at all, or at most only a very feeble inspiration, can be heard. During the expiratory efforts, too, very little respiratory sound is audible, and scarcely more than the sensation of a series of impulses can be perceived.

The total duration of the paroxysmal stage is exceedingly variable. In general terms it may be given as from three to six weeks, but it may last a shorter or a much longer time than this.

3. *Terminal Stage*.—The second stage merges so gradually into the succeeding one that no exact boundary between them can be recognized. The third stage may be said to begin when the severity of the disease is clearly diminishing. The attacks now grow less frequent and less severe; the whooping and vomiting persist for a time, but gradually disappear; and the cough, although still paroxysmal, grows distinctly looser and of a more catarrhal nature, and finally assumes the character of that of simple bronchitis. Hæmorrhages occur much less frequently, if at all; the bronchial secretion is now more muco-purulent, and the general health, if previously affected, improves. Finally the cough disappears entirely and the disease is over.

The duration of this stage is very variable. It may last from about ten days up to several months, depending upon hygienic and other conditions. Thus the approach of the winter season is liable to prolong it indefinitely. Not infrequently, after all cough has ceased and the child has appeared well, the development of a nasal or bronchial catarrh may be attended by a return of the paroxysms. Such a return cannot, however, be properly designated a part of the third stage.

Complications and Sequelæ.—Of the very numerous complications of pertussis those connected with the respiratory tract are most prominent. Bronchitis may be so in excess of the degree of catarrh usually present that it constitutes a complication. This is not an infrequent occurrence. Atelectasis very often develops in young children. It may affect only a small part of the lung or may be more extensive and threaten life, and is especially apt to be witnessed in weakly and rachitic children. Widespread broncho-pneumonia is one of the most common and most dangerous complications of whooping-cough. It usually comes as a result of atelectasis, but sometimes independently of it, and tends to run a very tedious course. As it develops the paroxysmal nature of the cough is very liable to diminish or disappear. Like atelectasis it is particularly prone to be seen in weakly children or when measles has immediately preceded pertussis, or in children who have been subjected to improper hygiene, especially exposure to cold. Pleural effusion,

empyema and croupous pneumonia are of less frequent occurrence; pneumothorax is rare; emphysema is common, but is generally only temporary. Sometimes, however, it is permanent throughout more or less of the lungs. Emphysema of the subcutaneous connective tissue has been reported but is very uncommon. Œdema of the glottis is sometimes seen. The coexistence of pseudo-membranous laryngitis is to be regarded as accidental.

A complication so frequent that it almost deserves to be called a symptom is the occurrence of a superficial yellowish-gray ulceration over or at the sides of the frænum of the tongue. It is probably produced by the forcible impulse of the tongue against the lower incisor teeth during the act of coughing. It has occasionally been seen in other disorders than whooping-cough.

Vomiting is generally to be regarded as a symptom of the disease, but the irritability of the stomach may become so great that it constitutes a genuine and very troublesome complication. In such cases vomiting is very frequent and takes place after every slight cough. Loss of appetite, indigestion, and diarrhœa are common complications, the latter being of a somewhat chronic nature, with the evacuation of considerable mucus. Prolapse of the rectum may result from the violence of the cough, and hernia may be brought about in the same way.

Hæmorrhages from various parts of the body occur during the paroxysms. Bleeding from the nose and mouth is so frequent that it is to be included among the symptoms of the disease. Subconjunctival hæmorrhage is not uncommon. Bleeding from the ear is a rare complication and hæmorrhage from the lungs is also unusual. Hæmatemesis, in which the blood comes originally from the stomach and is not previously swallowed, is certainly exceptional. Hæmorrhage into the skin occasionally occurs. Hæmorrhage into the meninges or within the brain is not an unusual complication, and is doubtless the cause of many instances of convulsions and other cerebral symptoms.

Convulsions are a dangerous complication and are not infrequent, particularly in young subjects. A persistent spasm of the glottis may sometimes cause death. Hemiplegia, aphasia, sudden blindness and other evidences of cerebral disturbance may be occasional complications.

General œdema of the skin has sometimes complicated the disease. Acute nephritis has been quite often reported.

Whooping-cough may be associated with diphtheria, varicella, scarlatina, or, in fact, any of the infectious diseases, but particularly with measles. The latter combination especially renders the prognosis more unfavorable.

Rachitis, anæmia and other constitutional maladies may complicate pertussis and influence its course unfavorably, or they may develop as sequels to it. Tuberculosis is a sequel very liable to arise in those who are predisposed to it or whose general nutrition has greatly suffered during the first disease. Its usual seat is the bronchial and intestinal glands or in some of the patches of broncho-pneumonia, but from these foci a more or less widely-spread infection may start. Epilepsy, various paralyses, aphasia, blindness, deaf-mutism following rupture of the drum-membrane, disseminated sclerosis and other conditions have been reported as occasional sequels. Some of them are to be viewed as accidental merely.

Diagnosis.—In the early stages of the disease the diagnosis can seldom be made with any certainty. The absence or scarcity of physical signs in the lungs, combined with the very harassing cough, which is markedly worse at night, renders the case suspicious. This is especially true if whooping-cough be prevalent at the time, or if there be a history of exposure to contagion. If the cough assume a decidedly paroxysmal character, the diagnosis becomes still

more probable. The occurrence of the whoop is usually conclusive, and even in those cases where this at no time develops, the nature of the cough, with such attending symptoms as vomiting, injection of the conjunctivæ and the like, makes the diagnosis fairly easy.

Severe acute bronchitis of the smaller tubes may sometimes be attended by a very spasmodic cough and may simulate pertussis closely; but the presence of numerous râles, with decided fever and dyspnœa, and the absence of more than a slight whoop will aid in distinguishing it. The same difficulty in diagnosis, and for similar reasons, may exist in cases where pertussis closely follows measles, since the severe bronchitis already present may appear to account fully for the severity, and even the paroxysmal nature, of the cough. The development of broncho-pneumonia during the first stage of pertussis may render the later diagnosis very difficult, since it is apt to modify greatly the character of the cough or even to prevent entirely the occurrence of the whoop. Tuberculosis of the bronchial glands may produce a paroxysmal cough much resembling that of pertussis. It is to be distinguished by a history of previous wasting and ill-health, the chronic course without distinct stages, the imperfect development of the paroxysms, which are unattended by abundant mucous expectoration or vomiting, and the presence of fever. Sometimes evidences of tuberculosis of the lungs are also present. A prolonged third stage of pertussis may readily simulate pulmonary tuberculosis, and, indeed, it may be possible that the latter disease is developing as a sequel. Only the later course of the case can decide.

Prognosis and Mortality.—Although the prognosis is favorable in most cases, yet pertussis is a far more dangerous disease than is ordinarily supposed. In England and Wales 120,000 persons died of it between the years 1858 and 1867, and 85,000 succumbed in Prussia between 1875 and 1880. Dolan ranks it third among the fatal diseases of childhood in England, and says it causes one-fourth of the annual mortality among children in London. Smith estimates that during fifty years there were 4840 deaths from it in New York City, or 1 in every 76 deaths from any cause. The relative mortality, as compared with the number of cases of the disease, is also larger than is commonly believed. Statistics vary regarding it, but it may be said to range from 3 to 15 per cent.

It is upon the great frequency of the complications that the high rate of mortality depends, for, if uncomplicated, the disease is not often dangerous. The younger the child the more unfavorable is the prognosis. The mortality is very much greater under two to three years of age than after this period, while after the fifth year it is trifling. The prognosis is rather more unfavorable in females than in males, owing possibly to a less degree of strength of constitution possessed by the former. The patient's previous general condition and the amount of care received while sick affect the prognosis very materially. The children of the poor, badly nourished and neglected as they so frequently are, are consequently apt to suffer most. Rachitis or any other constitutional debilitating disorder influences the course of the disease unfavorably. The presence of the winter season increases the danger through the greater liability of respiratory complications. On the other hand, the heat of summer brings on debilitating intestinal disorders. As already stated, convulsions and broncho-pneumonia are frequent and dangerous complications and the cause of many deaths.

Many cases pass safely through the attack, but die from the sequelæ. Some become marasmatic and die without the exact cause being discovered, although many of these are undoubtedly tubercular. Other cases show definite symptoms of tuberculosis of various parts of the body.

Treatment.—Prophylaxis.—In view of the highly contagious nature of the disease prophylactic treatment should be carefully carried out. Children who have not yet suffered from it should be rigidly kept from the slightest intercourse with those who are even suspected of being in the first stage of the malady. Inasmuch as there exists the greatest possible carelessness on the part of parents of the sick regarding the danger to others, it is better that uninfected children be removed entirely from the neighborhood whenever feasible. Particularly is this true in the case of delicate infants.

How long the danger of infection continues and how long quarantine must be maintained are not absolutely certain. It is admitted that the infectiousness diminishes during the third stage, and it may be assumed that by the end of two months after the onset of the disease the danger has entirely ceased. A still better criterion, however, is the entire cessation of the cough.

If, after the child has been apparently entirely well for a brief period, the cough, with or without the whoop, returns, it is probably safe to consider that the risk of infection is over in spite of this. It often happens that the whoop will thus return at intervals during months, or even for a year, whenever slight bronchitis is contracted. Quarantine during this entire period is manifestly unnecessary and impossible. The same is true of those cases which continue to whoop once or twice a day for an indefinite time. In such we may consider that after two, or at most three, months the disease itself is over, and that simply a neurosis remains: the "habit," so to speak, of whooping persists.

Although whooping-cough seems in nearly every instance to be communicated by the breath only, yet, to avoid the possibility of transmission in other ways, disinfection of the clothing, bed-linen, and the like should be carried out systematically, and the rooms used should receive a final disinfection before being inhabited by other children.

Treatment of the Attack.—The hygienic treatment of pertussis is of the utmost importance. Inasmuch as air loaded with carbonic dioxide has been proven to bring on paroxysms of cough, children should be kept in fresh air as much as possible. At the same time the very great sensitiveness of the respiratory mucous membrane must be borne in mind, and all possibility of taking cold must be avoided. In winter, therefore, it is often best to confine the patient to the house except on dry and still days. Where possible it is well to utilize two airy rooms, one of which shall be thoroughly ventilated and then warmed while the other is in use. The child can be changed from one to the other several times a day. The clothing should be warm enough to prevent chilling and consequent taking cold. The food should be nutritious, easy of digestion and assimilation, and frequently administered in cases where vomiting is a prominent symptom. In some cases of this kind it may be necessary to employ nutrient enemata.

It sometimes happens that change of climate will act most favorably upon the course of a case of pertussis. This is particularly true of the third stage if unusually prolonged.

The host of remedies recommended for pertussis is proof in itself that none of them constitute an infallible cure. Rather, however, than decri all medication, as is the habit with some, we should remember that negative results in the hands of one physician cannot vitiate positive results with any certain method of treatment in the hands of another competent observer. Nothing is more certain than that, although no medication is curative in all instances, many different methods of treatment are of undoubted value in different cases. Where, therefore, we fail with one, another must be tried in the effort to discover the remedy useful for the particular case. It must also be borne in mind

that to test the value of a remedy we must give it in sufficiently large dose, and further that it must be administered at the height of the disease, and not when the third stage has already commenced, at which time almost anything may seem to do good.

In the mild cases, where paroxysms are but few and of little severity, it is best to omit all medication intended to control the disease, and simply to keep a careful supervision over the patient. In severer cases, however, treatment is demanded. The condition existing in each individual case,—and, to a less extent, the stage of the disease—will exert an influence upon the choice of drugs to be employed. During the first stage, when the cough is hard and tight, with little expectoration and without full development of the paroxysmal character, the medicines to be selected are those useful in an ordinary bronchial catarrh. The same plan of treatment may be needed in the second stage, while in other cases the copious expectoration permits the freer use of sedatives. But inasmuch as the cough from the outset does not depend upon a simple bronchial catarrh, it is oftener better to begin the employment of remedies directed against the peculiar nervous character of the disease as early in the case as the diagnosis can be made. This need not interfere with any symptomatic treatment indicated. When the third stage is well under way attention must be paid principally to the accompanying bronchitis. Stimulating liniments to the chest may be useful, and tonic remedies are often demanded.

An attempt to consider all the drugs which have been employed for the treatment of pertussis would be so much a waste of time and space that only the most important of them can be mentioned here. Belladonna is one of those best and longest known and most widely used. Sometimes doses of moderate size suffice, but in other cases it is necessary to give it in increasing amounts until constitutional effects are seen. It often does great good, and often, too, entirely fails to relieve. The initial dose for a child of two years may be two minims of the tincture or one-twelfth of a grain of the extract three or four times a day. Alum is sometimes of distinct benefit, particularly when the abundance of the secretion appears to be the cause of frequent paroxysms. It may be given in doses of two grains every three or four hours at two years of age. It may sometimes be combined advantageously with belladonna. Quinine has been widely used with varying results. On the whole, it may be considered a useful remedy. When given internally the doses should be rather large—as one grain every two to four hours at two years of age—to produce an effect upon the disease; but there is risk of disturbing the digestion with it. It may be administered with advantage in suppositories, or, if by the mouth, disguised in syrup of yerba santa or syrup of licorice. Chloral is often useful to produce sleep at night. Two to four grains may be given at bed-time to a child two years old. There is some evidence that, administered at intervals during the day, it exerts also a direct influence upon the course of the disease. It can be exhibited either by the mouth or by enema. Its power of depressing must not be forgotten. Opium is frequently of the greatest service in obtaining temporary relief. Comparatively restful nights can often be procured by means of its administration at bed-time. It should, however, be reserved for the severest cases. Bromide of potassium or of some other base has been much recommended, and is often of distinct value. It lessens the nervous irritability, and in this way diminishes the frequency and intensity of the paroxysms. Its administration should be started immediately if evidence of nervous disturbance indicate impending convulsions. The dose at two years of age may be two to five grains, repeated according to the demands of the case. It may often be advantageously combined with belladonna. Cannabis

Indica has been much used, and is probably one of the most reliable means of treatment. Asafoetida is still a favorite with many. Carbolic acid, in doses of one minim at two years of age, has been found of service in many instances, but its toxic properties must not be forgotten. Peroxide of hydrogen has been highly praised, as have terpene hydrate and infusion of wild thyme. Ouabaine has been highly recommended. The dose is one-thousandth of a grain every three hours at five years of age. It is a powerful respiratory paralyzer.

Among the most important of other drugs which have been recommended for internal administration, and which have doubtless proved of service in some cases, are pilocarpine, lobelia, resorcin, grindelia, castania, drosera, camphor, quebracho, hyoscine, turpentine, benzole, carbonate of iron, and conium.

Antipyrine, first recommended by Sonnenberger, has been used with excellent results by so many that its value in the disease is now beyond question. Although, like other remedies, it often fails to relieve, many of the reported failures with it are doubtless due to the fact that it was not given in sufficiently large dose. Children bear it surprisingly well, and bad results following its administration are rare. The initial dose should be small, and the amount gradually increased until a child two years old receives one to two grains, or even more, every three hours. In a desperate case of pertussis in a four-months-old child under my care, in which three-quarters of a grain of antipyrine, given every three hours, failed entirely to relieve, an increase of the dose to one grain every three hours rapidly brought the patient from a condition of the greatest danger to one of comparative health. The child had suffered from very frequent and violent attacks of cough, followed by spasm of the glottis of so long duration that intense cyanosis with entire apnoea and loss of consciousness repeatedly resulted. Within forty-eight hours after the treatment had been instituted the little patient had passed an entire night and until afternoon on the next day with but a single paroxysm.

Phenacetin will sometimes be of service in cases where antipyrine has failed, and the reverse, of course, also holds good. Acetanilid has sometimes proved of use, but is less often employed and of less value than are its two cogeners.

Bromoform, one of the newest remedies for pertussis, was first recommended by Stepp in 1889, and has been largely used. It may be given in doses of from two to four drops three or four times a day at two years of age. It can be dropped upon moistened sugar or given in a mixture with alcohol, syrup, and water. My experience with it, although satisfactory to some extent, has not been as much so hitherto as published results had led me to hope. Some cases improved, but oftener small doses failed to be of service, while larger ones rendered the patient so sleepy and stupid that the remedy had to be abandoned. Nevertheless, the large number of reported cases in which the results have been extremely good indicate that the remedy is certainly of great value.

Local treatment of the respiratory mucous membrane has been largely employed. One of the most popular methods is the insufflation of quinine in the form of a fine powder. This may be applied directly to the larynx by the physician twice a day, or nasal insufflations may be made by the attendants several times daily. Excellent results have been obtained in each way. About one grain of quinine should be used at a time. Resorcin has been highly recommended by Moncorvo. A 1 per cent. solution may be applied to the pharynx and the opening of the larynx, or a powder may be insufflated into the nose, using one-half to one grain at a time for this purpose several times each day. The local application of a solution of cocaine has been advo-

cated, but is not without danger, as reported cases have shown. It has, however, often been of service in mitigating the severity of the disease. The solution should be of the strength of from 1 to 4 per cent.

With the steam or hand-ball atomizer the fauces and nares may be sprayed with the substances mentioned or with a weak solution of morphia. Bromide of potassium in solution is sometimes of much service, and tannin can be employed in the same way. Peroxide of hydrogen, in the dilution of one part in five, may be sprayed in the nares and upon the fauces, and very excellent results have been claimed for it.

Benzoin, boric acid, salicylic acid, iodoform, tannin, and other drugs, in powdered form, have found their supporters as useful agents for nasal insufflation. Benzoin is one of the best of them. Good effects can also be secured with boric acid.

Various volatile substances may be used with the atomizer in the form of vapor from boiling water. Carbolic acid is one of the best of these, and it is often of great advantage to allow the sick-room to be permeated by it. The action upon the cough is probably due in part to the anæsthetic effect of the carbolic acid, and largely to the influence of the moist atmosphere of the room, which loosens the mucus and facilitates its expectoration. Thymol, eucalyptol, and turpentine may be vaporized in a similar way. Chloroform and ether have been recommended for their general anæsthetic effect.

Remarkable results have been reported from the fumigation of the sick-room by burning sulphur. The child is to be washed in the morning, dressed in clean clothes, and placed in another room. The night-room is in the mean time thoroughly fumigated with the sulphurous vapor, closed during five hours, and then aired. The patient sleeps in this room at night. A single employment of this procedure has been effective in some cases.

The inhalation of the air in the purifying-rooms of gas-works is a method of treatment formerly much in vogue. The employment of the pneumatic cabinet has likewise been recommended. The use of the constant electric current has been advocated by several clinicians. The routine administration of emetics, once a popular procedure, is no longer in favor.

Complications demand, of course, treatment applicable to them individually.

TYPHOID FEVER.

BY F. GORDON MORRILL, M. D.,

BOSTON.

Synonyms.—Enteric fever; Slow fever; Fall fever; Gastric fever; Infantile remittent fever.

Definition.—An acute, infectious, continued fever, due to a specific cause, and characterized by prostration, wasting, enlargement of the spleen, inflammation of Peyer's patches and the solitary follicles of the intestine, and an eruption of rose-colored spots, which disappear on pressure being applied, and return rather slowly when it is removed. In children the solitary follicles rarely ulcerate, the eruption may be absent, and it is sometimes impossible to demonstrate enlargement of the spleen. The word "typhoid," first suggested by Louis on account of the supposed resemblance of the disease to typhus, has met with general acceptance in America and England, while in France the term "*dothiéntérie*" is frequently used by those who object to "typhoid" as misleading. "Enteric fever" is perhaps preferable, as suggesting the specific lesions of the disease, and is frequently employed as a substitute for the original name by precisians or by medical writers for the purpose of avoiding constant repetition.

History.—Previous to 1840 it was believed that children were exempt from typhoid, although good descriptions of cases (some with autopsies) had been published by Abercrombie, West, and others. During that year, however, Rilliet and Taupin published results of separate and independent investigations of enteric fever in children, and the fact of their susceptibility to the disease has since then become generally recognized. Later on it was proved that while typhoid is rare in infancy, it may occur in children at any age. Even so close an observer as Bouchut denied in 1867 that the disease ever occurred during the first year of life; but as a matter of fact the specific micro-organism of typhoid has been found in the liver and spleen of an infant who breathed only twelve hours, and whose birth took place during the fourth week of the disease in the mother; and in similar instances the specific intestinal lesions have been discovered. So it may be stated that, in childhood at least, no age is exempt.

Etiology.—As to the age at which children are most susceptible to the infection, statistics vary, but the risk probably increases from birth up to the tenth year, and then remains about the same until puberty is attained. The influence of sex is not apparent, although more boys than girls find their way into hospitals. The distribution of the disease is quite impartial, no climate being exempt. In America it is everywhere the prevailing fever. The influence of season is very marked, a large majority of cases occurring during the late summer and early autumn months. A dry hot summer increases the prevalence of typhoid—a fact which Pettenkofer attributes to the more thorough drainage of the soil into wells and springs, which are low,

and the water of which is, of course, concentrated; while Baumgarten suggests that at such times the poison is more easily disseminated in the air. Neither of these explanations is quite satisfactory, while each contains an element of truth.

Family predisposition to contract the disease is not infrequently observed. A marked instance of this susceptibility is cited by the late Charles Warrington Earle (in his article on typhoid fever published in the first edition of this book), where seven persons of one family contracted enteric fever by visiting an infected room or nursing other cases so caused. As a rule, the previous condition of health plays but an insignificant part in the etiology of typhoid, which is directly caused by absorption from the alimentary canal of the specific micro-organism (named after its discoverer, Eberth), which is a short, thick bacillus with rounded ends and containing glistening spots which remain unstained when subjected to the ordinary process. It occurs singly or in chains, and its appearance varies in accordance with the medium in which it is grown. The variety of ways by which different authorities say it can be distinguished from the *bacillus coli communis* is suggestive of the fact that there is a great liability to error; and in this connection it is proper to state that it is claimed that the Eberth bacillus has been found in the fecal evacuations of persons free from any suspicion of typhoid, and who had never had the disease. That the bacillus is often swallowed with impunity is undoubtedly true—the soil must suit the seed, as in other infections. Whether the Eberth bacillus can remain inactive in the alimentary canal for any considerable length of time, and then suddenly cause disease (as does the *Klebs-Löffler* bacillus in the throat and nose), remains to be proved. Be this as it may, the poison finds entrance to the body through the nose or mouth, and usually in articles of food or drink.

Water that has been contaminated by the discharges of those having the disease is by far the commonest source of infection. Examples of this contamination through cess-pools, drains, and the washing of excreta for a considerable distance into streams and reservoirs are too well known to bear repetition here. In Paris the river Seine has a firmly established reputation as a conveyer of the enteric bacillus. When the usual sources of supply for certain quarters of the city fail, Seine water is substituted, and an epidemic of typhoid follows with unfailing regularity in the course of two or three weeks from the time when it is turned on. The bacillus grows rapidly in fresh milk, which is a frequent source of infection, and is sometimes responsible for outbreaks confined, in the main, to children. Washing the cans in infected water is the usual explanation of the contamination. Any article of food or drink may be infected by the person having the disease, or, indirectly, through carelessness on the part of the attendants. Oysters may absorb the micro-organism from drainage, the bacillus retaining its characteristics perfectly well after a fortnight's sojourn in sea-water. Freezing does not destroy its vitality, and ice may thus act as a carrier of the disease.

In view of the infinite variety of ways (food, drink, bedding, toys, books, utensils of all sorts, and probably the air we breathe) in which the bacillus, moist or dry, may be distributed, it is a matter of surprise that the disease is not even more prevalent, as it doubtless would be if every one swallowing the poison were susceptible.

After entering the alimentary canal, the micro-organism penetrates the mucous membrane and gives rise to profound constitutional disturbance, together with characteristic changes in the intestines and other organs. The

length of time which may elapse after exposure before the symptoms manifest themselves varies within wide limits. It is fixed by the Clinical Society as "eight to fourteen, sometimes twenty-four, days." Liberal as this rule is, there are well-marked exceptions to it. In a recent epidemic near Boston two children were taken obviously sick, with what proved to be typhoid fever, forty-eight hours after drinking for the first and only time infected milk, to which the source of trouble was clearly traced. In other instances five days covered the period of incubation in children, and a somewhat longer period in adults of the same families.

Morbid Anatomy.—The post-mortem appearances which enteric fever causes in adults will be mentioned only for the purpose of contrasting them with lesions of the same organs as observed in children.

Rose spots usually disappear after death, while accidental eruptions (sudamina, etc.) persist.

The duodenum may be slightly congested, while the changes in the jejunum and ileum are usually due to hyperplasia, and not (as in adults) to ulceration. Peyer's patches and the solitary follicles are surrounded by zones of congestion, but induration is rarely perceptible to the touch; in other words, the congestion is not sufficiently intense to interfere seriously with the blood-supply, and for this reason ulceration, except to a slight degree, is seldom present. Whatever the intestinal lesions may be, they are seen in greatest number in the immediate vicinity of the ileo-cæcal valve.

According to the combined statistics of Pfeiffer and Montmollin, lesions of the intestinal mucous membrane, varying from the (usual) superficial congestion to deep ulceration with perforation, were present in 72 per cent. of their cases.¹

Ulcerations, when seen, rarely exceed ten or twelve in number, and their superficial character contrasts strongly with similar lesions in adults, which so frequently involve the submucosa, and may be so confluent in the neighborhood of the ileo-cæcal valve as to form an eschar of great size. Instances of deep ulceration are rare in children, but when present are due to the same process as in adults, which reaches its height in eight or ten days, and then undergoes a retrograde change or produces necrosis. Retrogression is fortunately the rule in children, and ulceration seldom reaches the muscular coat of the intestine, which in adults usually constitutes the floor. Perforation is very rare, but does occur. As a rule, the solitary follicles do not ulcerate: they are swollen and often present the appearance described by French writers—a beard of two days' growth. In rare instances they ulcerate, and I find in the records of the Boston Children's Hospital one case in which this lesion was present in the solitary follicles of the cæcum, extending several inches below the valve.

The mesenteric glands are swollen, particularly in the vicinity of the ileo-cæcal valve, and the intensity of this condition does not necessarily correspond to the extent of the intestinal lesions. Peritonitis, with or (rarely) without perforation, is observed very exceptionally. The spleen is certainly of normal size in some cases, but, as a very general rule, is swollen and hyperæmic. If death occurs at a late stage of the disease, it may be soft, and has been known to fracture (*ante-mortem*) on palpation. Hæmorrhagic infarcts are common. The liver may be hyperæmic and enlarged in severe cases, or it may be soft and the bile colorless; but, as a rule, hepatic lesions are slight and insignificant as compared with those of adults.

¹ It must be borne in mind that this estimate applies to fatal cases, in which intestinal lesions are naturally much more frequent and serious than in those who survive.—F. G. M.

The brain is singularly free from important pathological changes, and even in cases where nervous symptoms have been decidedly marked, nothing beyond a congestion of the pia mater and (to a slighter degree) of the brain-substance, together with extremely moderate distention of the arachnoid, is observed. The heart is pale, and often softened by granular or fatty degeneration of its muscular fibres. Passive congestion of the lungs is common, and patches of broncho-pneumonia of the deglutition type are not rare. The kidneys may show signs of granular degeneration, but rarely of true nephritis. The voluntary muscles, particularly the pectorals, recti abdominis, and adductors of the thighs, may be in the same condition as those of the heart just described. This of course may be the case after any prolonged sickness, and is not peculiar to typhoid fever.

Ulcerations of the laryngeal cartilages, periostitis, osseous necrosis, and suppurative parotitis are extremely rare, but have all been observed. In the case which I have referred to as appearing in the Children's Hospital records Eberth's bacillus was found in the lungs and in great abundance in the spleen, liver, and kidneys.

Symptoms.—After a variable time from the date of exposure the child begins to lose its interest in play, shows signs of lassitude, and is inclined to lie down. Headache, anorexia, chills or chilly sensations, nausea, epistaxis, pain in the back or legs, diarrhoea (or constipation) may be present. This condition of things may continue for a week, or even longer, before the child takes to bed and is obviously sick. More rarely the onset is sudden and accompanied with vomiting. In either case, in the absence of any suspicion of typhoid infection, the patient's condition often passes as the result of indigestion or having "taken cold." But the usual remedies fail to give relief—the symptoms persist, and are so marked as to make it evident that no temporary indisposition can account for them satisfactorily. The arbitrary date of the commencement of the "run" of the fever is now fixed.

The degree of constitutional disturbance which typhoid gives rise to in children is usually much less than that which it causes in adults; but it is sufficiently well marked, as a rule (in America, at least, where the abortive and extremely mild types are comparatively rare), by the end of a week to enable one to make a diagnosis. The child lies with flushed cheeks and an expression of marked apathy, which remains present until the fever subsides, and occasionally for days after the temperature has become normal. The abdomen, flat at first, becomes swollen and tender on pressure, particularly in the right iliac fossa. Sometimes abdominal pain is voluntarily complained of. The spleen is apt to be swollen, and its lower edge can be felt (usually below or under the false ribs, but occasionally more toward the front) in a majority of cases. Rose spots may be visible on the abdomen, the lower portion of the thorax, the inner surfaces of the thighs, or between the shoulder-blades. A moderate diarrhoea may be present, but constipation is more frequently the rule during the first week after the child comes under observation. The urine is scanty and high-colored. Bronchitis or, rather, cough, is not uncommon. The skin is usually dry and hot, but perspiration is exceptionally observed during the early stage.

The lips are dry and scaly. Sordes may collect on the teeth and gums if care is not observed. There is no characteristic appearance of the tongue, which is almost always moist, red on the tip and along the edges, and coated with a yellow deposit which is variable in thickness and distribution, sometimes covering the entire upper surface, or being confined to the anterior half or to the lateral portions only. Anorexia is complete, but the

child takes kindly to cool liquids. Sleep is apt to be disturbed, and mild delirium is not uncommon during the night. The pulse beats from 120 to 140 per minute, and the temperature reaches 104° to 105° F. (oftener the former) at night, with morning remissions of 1.5° to 3° F. As the disease progresses emaciation becomes marked. Diarrhoea and abdominal pain, which may precede or follow the loose discharges, are common, but constipation may continue until the case terminates. Attacks of nausea lasting two or three days may occur. Prostration and apathy are more profound, and there may be retention of urine.

Toward the end of the second week of the child's confinement to bed in mild cases, or a few days later in those of average severity, the temperature begins to descend by lysis (often preceded by very marked morning remissions), and soon reaches the normal point. Convalescence now begins: the appetite becomes ravenous, and, if no relapse occurs, complete recovery in all but the matter of physical strength soon follows. The anæmic pallor and weakness caused by enteric fever are very marked. The child's first attempts to walk with its attenuated legs bear testimony to the severe constitutional disturbance it has passed through. The hair falls out to a greater or less extent, and this, together with a perceptible increase in height (typhoid stimulates the growth of the long bones), causes the patient to present a curious aspect.

The usual features of an average case having now been roughly outlined, special symptoms and complications will be considered:

Relapse.—A recrudescence of fever from no apparent cause is not uncommon. It is apt to occur a very few days after the beginning of convalescence, and usually lasts a day or two only. True relapse, due to reinfection after a perceptible period of apparent convalescence, is usually of sudden onset, and occurs with varying frequency in different epidemics. At the Boston Children's Hospital 17 per cent. of 100 recorded cases have had a relapse on the (average) thirty-third day after the first symptoms of the original attack were noted. The mean duration of these relapses was seventeen days. Of those affected, 12 were girls and 5 were boys—a fact which corroborates, in a modest way, Montmollin's statement that the frequency of relapse is influenced by sex. As a rule, the relapse is neither so long nor so grave as the original fever, but occasionally it may be severe enough to cause death. A second relapse may occur. This happened in 4 of the 17 cases I have referred to, and all of them recovered. Instances of a third relapse have been recorded—the greatest number which I have seen mentioned in connection with the typhoid fever of childhood. Intercurrent relapses are not very uncommon, and an unusually prolonged pyrexia may often be accounted for in this way. The symptoms of relapse differ in degree only from those which the patient has already had.

Respiratory System.—Epistaxis is rather common, and of no importance save from a diagnostic standpoint. It was noted in 5 per cent. of 70 cases by Forchheimer, and in 20 per cent. of the 100 cases which I have mentioned. Cough is frequent, and is usually caused by slight bronchial catarrh or some ordinary affection of the upper respiratory tract: I find it noted in 36 per cent. Well-marked signs of bronchitis are somewhat rare. Bronchopneumonia (often of the deglutition variety) occurred in 7 per cent., and in 1 fatal case the Eberth bacillus was found in the inflamed lobules. Congestion of the bases is usual in prolonged cases, and would be even more common if children did not voluntarily change position far oftener than do adults. Frank pneumonia is extremely rare, although typhoid patients are by no

means proof against other infections. Ulceration of the vocal cords and necrosis of the laryngeal cartilages, with resulting stenosis, have been observed. The ordinary forms of sore throat are common enough, and diphtheria can be readily contracted during the course of enteric fever.

Digestive System.—The lips are dry and apt to crack if the child is allowed to pick at them. Herpetic eruptions are not common. The gums may be soft and swollen. The brown tongue so often observed in adults is seldom seen. The organ may be dry and red, but soreness is seldom present. The bowels are usually constipated at first, and diarrhoea is apt to come, if at all, during the second week. There may be seven or eight discharges in twenty-four hours, which may or may not be of the familiar "pea-soup" variety. This condition usually subsides rather slowly under appropriate treatment, but is apt to recur. Involuntary discharges are rare excepting in very young children. Abdominal pain on pressure increases during the second week. Intestinal hæmorrhage (as would be naturally expected from the rarity of deep ulceration) is seldom observed. It was noted in 4 per cent. of the 100 cases mentioned. In 2 of these it consisted of small quantities of blood passed with each evacuation for several days, and both recovered. In 1 instance it was slight, but the case was one of intense typhoid infection, with many lesions of the internal organs, and the bleeding caused death from exhaustion. An autopsy failed to reveal the vessel from which the blood had escaped, in spite of a very careful and prolonged search. In the fourth case two profuse hæmorrhages, which occurred within twenty-four hours, were speedily followed by perforation, peritonitis, and death. Perforation (said to be more common than hæmorrhage) is rare. Professor d'Espine (of Geneva) has seen but one case. It is apt to occur, if at all, at a late stage of the disease, and has been observed in one instance five weeks after the beginning of convalescence. Peritonitis without perforation has been observed by J. C. Wilson, J. Simon (of Paris), and other leading authorities, but is extremely rare. Usually it is the direct result of perforation, and if the rupture takes place at a point which is in contact with a solid viscus or a coil of intestine, the peritonitis may be limited and recovery follow. Otherwise the contents of the alimentary canal escape, and speedily cause acute general inflammation of the peritoneum (accompanied by pallor, clammy sweats, abdominal distention, small and frequent pulse), which proves quickly fatal. Enlargement and suppuration of the parotid gland have been observed by various authorities.

The Skin.—Rose spots, if present, usually make their appearance within a week after the disease is fairly established. As a rule, they are not so well marked in children as in adults, and are less common and numerous in America than in Europe, where an abundant eruption is regarded as a good omen. Ashby and Wright state that they are absent in only 25 per cent. of all (English) cases. I find them noted in 53 per cent. on the (average) twelfth day after the first appearance of any symptoms of the disease. In rare instances they are seen during a relapse, when careful daily investigation has failed to discover them during the original attack. Furunculosis may occur at a late stage or during convalescence. Sudamina and eruptions resembling rose spots, but failing to disappear under pressure, are common—more particularly the latter. The nails become fissured transversely from temporary cessation of growth. Wilson mentions a faint diffuse erythema of the legs during the first week. Acute otitis media with perforation (unless relieved by incision) occurs in a certain percentage of cases, and this may or may not influence the range of the temperature. In 40 cases which entered my wards

during the fall of 1896, it was observed 5 times. Bed-sores are easily avoided, except in the severest cases.

The Spleen.—It is probable that the spleen is enlarged to some extent in all cases at some period of the disease, although this cannot always be demonstrated by percussion or palpation. The fact that this organ has been found to be of normal size in a few cases which have been autopsied is no proof that it had not been enlarged during the acute stage of the fever. To palpate the spleen the child is made to lie upon its right side, with the knees flexed and drawn up, and the fingers are gently but firmly pushed upward under the false ribs; then, if the patient can be induced to take a deep breath, the lower edge can often be felt. Percussion of the organ, unless the results are corroborated by palpation, is not satisfactory. In 40 recent cases at the Children's Hospital the spleen was palpable in 23. The enlargement usually disappears very soon after the temperature becomes normal. If it remains, relapse may be expected. Splenic enlargement is of course not peculiar to enteric fever, but may be present in any infectious disease. Bartholow cites a case of rupture of the organ from slight violence, and the fact that at autopsies it has been sometimes found to be a mere bag of pulp shows the possibility of such an accident being caused by too vigorous efforts to detect a symptom which is rarely essential to enable one to distinguish typhoid fever from other diseases. Hepatic enlargement is very seldom of sufficient extent to be noteworthy.

The Urine.—Ehrlich's diazo reaction, a description of which is hardly needed here, has been found present in 136 of 196 cases of enteric fever (Osler). Its diagnostic value is much impaired by the fact that it is not infrequently seen in other acute febrile affections. In 50 selected cases Dr. J. Bergen Ogden of Boston found that the reaction was present between the (average) fourteenth and twentieth days of the disease, and remained so for from six to eight days.

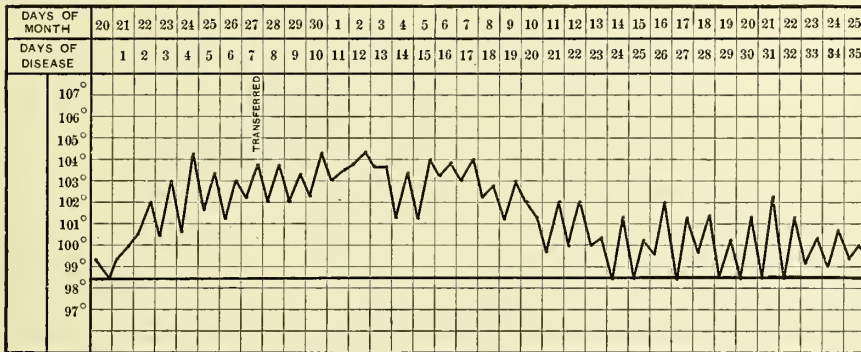
Nervous System.—Complete indifference to surroundings is the rule, and delirium, if present, is usually of a mild and harmless type. Occasionally a child will try to get out of bed, and is somewhat difficult to manage, and mechanical restraint is required in rare instances. Mild delirium, associated perhaps with night-terrors, is not at all unusual, and is easily controlled by appropriate means. Trembling of the hands and twitching of the facial muscles are rare. I have seen this in the form of a one-sided affection, and the movements resembled those of chorea. Retention of urine is less common than in adults. Hyperæsthesia of the lower extremities and pain in the feet and ankles are sometimes observed, but any marked degree of peripheral neuritis is extremely rare; and the same may be said of cerebral meningitis. Ominous brain-symptoms (active delirium, intense cephalalgia, strabismus, vomiting, and retraction of the head) have been known to disappear in a few days. Mental disturbances (delusions, melancholia, etc.), which appear in exceptional cases during the course of enteric fever, sometimes continue long after convalescence has been established, but they tend to disappear as the child's strength becomes restored, and seldom last more than a few weeks. Transitory aphasia and hemiplegia have been noted at a late period of the disease. It may be said, in a general way, that all nervous symptoms occurring during typhoid in children are likely to disappear in time.

The Heart and Pulse.—Slight myocarditis with a feeble apex-beat and softened first sound, accompanied by a feeble and perhaps dirotic pulse, are common. In severe cases the pulse intermits or becomes irregular, and in those in which the condition of the heart is the direct cause of death the

sounds may assume a foetal rhythm, which precedes a fatal termination for a day or two only. The average rate of the pulse is from 120 to 150, and its curve quite closely follows that of the temperature on the chart. A slow pulse with a high temperature is occasionally observed for a day or two, but the reverse is extremely rare. In 3 of the cases which I have mentioned a pulse of 180 was recorded, and 2 of them proved fatal. Endocarditis and pericarditis are seldom seen.

Temperature.—It is said that the temperature during the initial stage lacks the characteristics which are of such essential aid to the diagnosis of

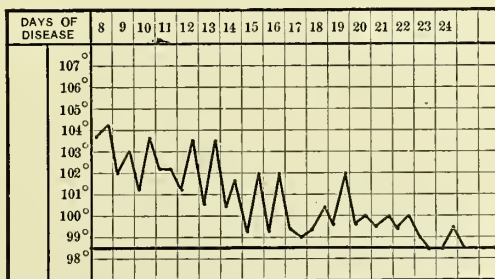
FIG. 1.



Showing temperature of initial stage. (Boy aged 5 years.)

enteric fever in adults, but an instance in which an accurate record of the temperature was kept for several days before the diagnosis was made does not confirm this statement. As may be seen by reference to Fig. 1, the temperature rose steadily and reached 102° F. in forty-eight hours, when morning remissions promptly occurred, while the evening temperature continued to mount higher. The remissions average about 1.5° F. after the disease is

FIG. 2.

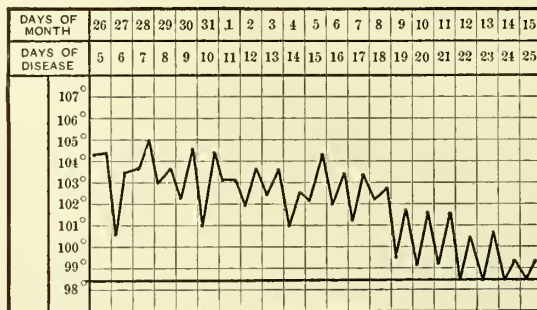


Showing marked morning remissions during the last days of a short case, also slight recrudescence of fever. (Boy aged 6 years.)

fairly established, and may be counted on with a considerable degree of certainty. During the few days preceding convalescence they often cover from 2° to 3° F., this corresponding (to a degree which the comparative insignificance of the intestinal lesions would lead one to expect) to the second stadium as seen in adults. During this short stage of marked remissions the morning temperature may be normal for two or three days before convales-

cence is attained, as shown in Figs. 2 and 3. Lysis is the general rule, but occasionally the termination is somewhat abrupt, as it is apt to be in the abortive cases of adults. The average highest temperature observed in 100 cases at the Children's Hospital was 104.5° F., and this was noted on the (average) twelfth day from the first appearance of symptoms. The extremes

FIG. 3.

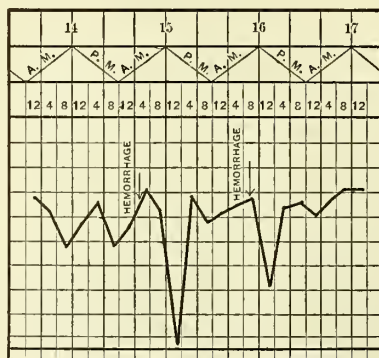


Showing morning remissions a few days before convalescence. (Boy aged 12 years.)

were 101° and 107.8° F., the latter case recovering. In 5 of 7 fatal cases a temperature of 105° F. or more was reached.

As regards the duration of the fever, a normal morning temperature was observed on the (average) twenty-fourth day, and a normal evening temperature on the (average) twenty-ninth day after the first appearance of symptoms. This of course applies to pyrexia as a symptom *per se*, and not to the child's general condition, convalescence being not infrequently well underway before an absolutely normal temperature could be recorded. A fall of temperature accompanies any considerable hæmorrhage. Fig. 4 shows the descent attending two evacuations of coffee-colored blood (at least eight ounces

FIG. 4.



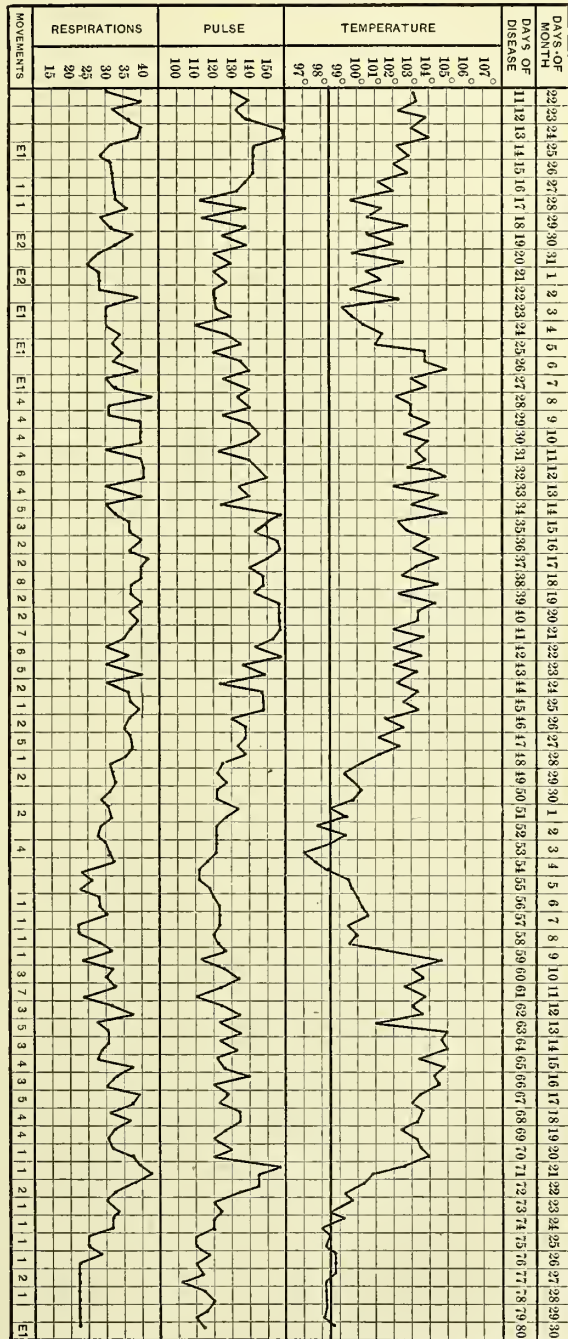
Showing sudden fall of temperature after each hæmorrhage. (Boy aged 10 years.)

each time) occurring on successive days. In one instance a sudden depression (6.8° F.) from no apparent cause was noted, and slow recovery followed. Examination of a number of four-hour charts of cases in which neither antipyretics nor cold baths were used shows that during the acute stage the lowest temperature is recorded at 8 A. M., and the highest from twelve to

fourteen hours later. A slight remission occurs after midday and midnight. Fig. 5 shows the temperature, pulse, and respiration of a case of double relapse, together with the number of evacuations daily, the patient eventually recovering.

Diagnosis.—It is usually a sufficiently easy matter to recognize enteric fever in a child when the disease has become fairly established, but during the first four or five days, in the absence of other cases in the neighborhood, it is frequently impossible. The symptoms may correspond to those caused by digestive troubles, or by some fancied exposure to "taking cold," or by ephemeral fever due to an unknown cause. In hospitals the patient is seldom seen until there is good evidence of serious illness. The diseases with which typhoid is most likely to be confounded are — tuberculous or epidemic meningitis, acute miliary tuberculosis without brain-symptoms, frank pneumonia, and malaria. Tuberculous meningitis is liable to occur in hospital patients under constant observation for disease of the hip or spine, and the records of cases of this kind show that night-cries, a well-marked *tâche cérébrale*, and inequality of the pupils (aside from the peculiarities of pulse and temperature) are the earliest signs which are of essential aid in making a differential diagnosis. Epistaxis and bronchial catarrh are of diagnostic value in favor of typhoid, while vomiting and headache are more persistent in tuberculous meningitis. The temperature of an established case of enteric fever

FIG. 5.



Showing respiration, pulse, and temperature of a case with double relapse. (Boy aged 8 years.)

differs from that of a beginning tuberculous meningitis, which is very irregular and seldom reaches 104° F. until unmistakable signs of brain-trouble are present. A very quick pulse with a low temperature is common enough in tuberculous meningitis, but rare in typhoid, in which disease the pulse follows quite closely the temperature-curve on the charts. Irregularity of the respiratory rhythm is sometimes observed in tuberculous meningitis. In any event, a tapping of the spinal arachnoid or an examination of the blood (to be spoken of later on) soon clears up cases which may remain doubtful in the absence of other well-marked diagnostic signs.

The onset of well-marked cerebro-spinal fever is sudden, and accompanied by intense cephalalgia, dilated or contracted pupils, which fail to respond to light, and retraction of the head and neck—symptoms rarely present in the typhoid of children, and almost never in the early stage of the disease. As a matter of fact, one is much more apt to mistake a “cerebral” frank pneumonia for cerebro-spinal meningitis than the latter for an enteric fever.

In acute general tuberculous infection the abdomen is usually flat, the temperature irregular, while the family history of the patient and the presence of enlarged superficial glands may aid in diagnosis. Bronchial catarrh is common to both miliary tuberculosis and typhoid, and, so far as the spleen is concerned, a considerable enlargement may be present in either. Rose spots, epistaxis, and splenic enlargement may all be absent in enteric fever, and the resemblance to general tuberculosis may be so close that only an examination of the blood can conclusively settle the question.

Malaria in children is very apt to be accompanied by quotidian (double tertian) paroxysms, which may cause it to be confounded with typhoid. But the absence of rose spots and abdominal tenderness, together with the effect of one fair-sized dose of quinine (administered immediately after a paroxysm), quickly decides a question which is otherwise easily answered by an examination of the blood.

Frank pneumonia may closely resemble enteric fever when the physical signs of consolidation fail (as they sometimes do) to develop for several days. The temperature of the two diseases is very similar (barring the usual irregularity of the morning remissions in pneumonia); abdominal pain is common in either; and in the absence of rose spots, abdominal tenderness, and enlargement of the spleen, Widal's blood-test may be required to enable one to reach a conclusion.

“Cerebral” pneumonia, as I have before remarked, is more likely to be confounded with epidemic meningitis than with typhoid; but apex-pneumonia may come and go with few if any signs pointing to pulmonary trouble. The evidence obtained by listening to the chest may be very indefinite—a mere suggestion of bronchial respiration and dulness, which vanish rapidly and require frequent examinations to detect.

Grippe is distinguished by a degree of prostration disproportionate to the other symptoms, the absence of the characteristic temperature of enteric fever, and the fact of its being epidemic. Very young children suffering with grippe are apt to be extremely irritable—a mental condition which contrasts strongly with the apathy usual in typhoid. In the early stage of either disease there may be fever, delirium, bronchial catarrh, muscular pains, and diarrhoea, while later on the absence of rose spots, enlargement of the spleen, and abdominal tenderness may render the differential diagnosis extremely difficult.

In all doubtful cases evidence which seems to be almost always reliable can be obtained by means of the test discovered and perfected by Pfeiffer,

Gruber, Durham, and Widal. This consists in adding one part of blood-serum from a suspected case to ten parts of a bouillon culture of typhoid bacilli. If the culture is fresh and the serum that of a person having enteric fever, a characteristic reaction takes place, which may be briefly described as a gradual loss of motility on the part of the bacilli after their aggregation into groups. This same reaction can be obtained from the milk of nursing women who may happen to have typhoid, and occasionally from the urine; but the latter is not reliable, as the same phenomena may be produced by the urine of healthy persons. The reaction can also be obtained with dry blood, a drop of which upon a folded piece of sterilized non-absorbent paper is examined "by moistening with a drop of sterilized water, mixing the solution with a drop of the bouillon culture, and examining the mixture as a hanging-drop preparation under a dry lens of medium power."¹ It is claimed that this method is less likely to give rise to confusion than the one in which serum is employed, unless the examination be made without delay.

The great convenience of the dried-blood test, as compared with that in which fresh serum must be used, makes it a subject for congratulation that its reliability has been established by Drs. Johnston and McTaggart. As a rule, they have found the reaction well marked and prompt after the fifth day of the disease. Samples of blood kept dry in the ordinary air and temperature of the laboratory for sixty days still gave a good reaction.

Prognosis.—The combined statistics of Baginsky, Steffen, Montmollin, Henoch, and Wolfberg give an average mortality of 7 per cent. Comby states that it is between 6 and 7 per cent. It is my impression that it is about the same in America as in Europe. At the Boston Children's Hospital it has been nearly 7 per cent. The above figures, taken in the main from results obtained in hospital practice, cannot accurately represent the mortality of all cases, many of which are so light as to pass unrecognized, and a certain proportion of which occur among children of the well-to-do, who are treated in their homes and whose previous nourishment and sanitary environment have been good. Perhaps 4 per cent. is a fairer estimate of the proportion of fatal cases. Special symptoms which point to a fatal termination are—pneumonia involving any considerable extent of lung, tuberculosis, previous poor health, intense pyrexia, marked weakness and irregularity of the heart, parotitis, considerable hæmorrhage, peritonitis from perforation, and symptoms of cerebral complications lasting more than a week. The intensity of infection must be considered, as well as the fact that a mild attack may kill a tuberculous or syphilitic child. Copious and obstinate diarrhœa and prolonged vomiting are unfavorable signs.

Treatment.—Adequate ventilation, liberal air-space, strict attention to the comfort and cleanliness of the patient, and the steady maintenance of a temperature of 65° to 70° F. are to be ensured. Children with typhoid fever have no appetite, as a rule, for solid food, but they are (fortunately) thirsty, and take cold milk with relish. Three- or four-ounce portions of milk (less to very young children) should be given every three hours. In this way a child five years old will take from eighteen to thirty ounces in twenty-four hours, and older ones in proportion up to two quarts, which is apparently the limit of their capacity. Should nausea or vomiting interfere, the milk should be diluted with Célestins Vichy, or lime-water, and given in very small but frequently repeated portions. As a rule, the stomach yields to this simple treatment within forty-eight hours, but if these measures do not

¹ Drs. Wyatt Johnston and D. D. McTaggart of Montreal, in the *American Medico-Surgical Bulletin*, Jan. 10, 1897.

suffice, it is best to withdraw the milk and substitute teaspoonful doses of egg-albumin-water with a few drops of brandy. In cases of considerable severity, where there are signs of prostration, brandy should always be used. A teaspoonful *ter in die* is often enough to regulate a weak pulse and contributes greatly to the child's comfort, but there should be no delay in increasing the amount if the patient fails to respond to this very moderate stimulation. In looking over the records of the Children's Hospital, I find but one case in which so much as three ounces was given for any length of time—this in the middle of a second relapse, from which the patient (a puny child five years old) completely recovered. There are but few children that will not derive benefit from moderate stimulation at some period of an attack of enteric fever. Cold water is often craved, and may be given quite freely if the amount of milk taken besides is sufficient to nourish the patient. During the acute stage the diet should be restricted to milk only, any change being liable to produce gastric or intestinal disturbance. When the temperature shows that lysis has begun, or when sharp morning remissions, together with the child's brighter aspect, signal the speedy advent of convalescence, some form of predigested starch and somatose may be safely given. The possibility of relapse must be borne in mind, and, whether the improvement is followed by uninterrupted recovery or merely preceded by a few days the occurrence of reinfection, an increase of nourishment is plainly indicated. After convalescence (which is tedious in the mildest cases) is fairly under way, the ravenous appetite may be satisfied with no untoward results, unless a slight recrudescence of fever (not a relapse) gives warning that the digestive powers are being overtaxed.

The Brand method, so far as I have been able to ascertain, has never been systematically employed in any large number of cases in children; but the marked reduction in the mortality of the disease attending this treatment in adults certainly warrants its thorough trial in cases where a sufficient number of competent attendants can be had to ensure its being properly carried out—a condition by no means easy to fulfil.

In the first stage, if constipation is present, calomel can safely be given, both as a purge and an intestinal disinfectant. Less than a grain (given in triturates of gr. $\frac{1}{10}$ every hour) is usually enough to produce one or two free evacuations. If diarrhoea is present when the patient has been ill but a short time, calomel may still be used in the same way before employing drugs to check the trouble. Of all intestinal antiseptics for continuous use (and the diarrhoea of enteric fever yields but slowly to treatment, as a rule), salicylate of bismuth gives as satisfactory results, perhaps, as any. Given in five- or ten-grain doses, *ter in die* (and an additional dose during the night if the trouble persists and disturbs the child's sleep), it usually modifies the number and character of the evacuations in a few days, and, should the same condition recur (as it often will), there is no apparent advantage gained by changing the treatment, so far as I have observed. Cool bathing will reduce a high temperature, but the relief thus obtained is slight (a descent of 1–2° F.), and so temporary as to hardly compensate for the trouble involved. Lactophenin and pepsol (gr. 3–8) in divided doses are very effective antipyretics, and perfectly safe unless there is some obvious contraindication to their use. The former is not quite so effective as the latter, which will cause an average reduction of 4° F. three hours after its administration. Quiet sleep may often be obtained in this way where the temperature is high, and no harm result, as far as I have been able to observe, from employing either drug in suitable cases. If insomnia is a marked feature of a case in which the condition of the patient does not warrant the exhibition of antipyretics, trional

in five-grain doses is indicated. Very moderate doses (mij-v) of digitalis are most effective in regulating a weak or irregular pulse when brandy fails to accomplish the purpose.

In ordinary cases the drugs that I have mentioned will fulfil all the usual indications for interference with the natural course of a disease which, fortunately, tends to recovery. Hæmorrhage, perforation, organic brain-trouble, and the overwhelming intensity of the infection, as seen in typhoid fever of adults, are rare in children, and hence the treatment is comparatively simple. Hæmorrhage is the most frequent complication that demands immediate and active treatment, and in case of any considerable bleeding the foot of the bed should be raised, ice-bags applied to the abdomen, and astringent remedies (gallic acid or a combination of lead and opium) given, together with ergotin by hypodermatic injection. Perforation, if in a position to cause general peritonitis, is speedily fatal without surgical aid, which should be instantly obtained, and the results of which are thus far very encouraging, as shown by the statistics of Drs. W. W. Keen and Thompson S. Westcott of Philadelphia—83 operations with 19.36 per cent. of recoveries. Five of the cases operated upon were children, two of whom were saved.¹

Bed-sores are easily avoided by strict attention to keeping the child dry and clean. Sordes are prevented by a little care on the part of the attendants. Acute active delirium is rare, but forcible restraint is occasionally required to prevent a child from getting out of bed. Mental disturbances, which persist after convalescence is reached, almost invariably disappear without special advice or treatment. Ominous symptoms of cerebral trouble occurring during the acute stage often vanish so quickly as to preclude the possibility of their being due to organic lesions. I have the notes of a case in which a convergent strabismus, delirium, somnolence, and a *tâche cérébrale* disappeared twenty-four hours after they were noted. The application of ice-bags to the head and an increase of stimulation are usually indicated when nervous symptoms predominate.

Prophylaxis.—All soiled diapers and sheets are to be at once removed and allowed to soak for six hours in a 1 : 40 solution of carbolic acid, and then boiled and washed in vessels devoted especially to this purpose. The nates must be carefully wiped with cloths dampened with a 1 : 40 solution. These should be burned or treated in the same manner as the diapers after being once used. Discharges which are received in bed-pans are to be covered with a 1 : 20 solution of carbolic acid or with thin whitewash, and, after any solid fragments have been thoroughly broken up, should be allowed to stand twenty minutes before being emptied into the hopper, which must be kept scrupulously clean. Rubber covers should be provided for the bed, and washed off with the 1 : 40 carbolic solution. Cups, glasses, spoons, and feeding utensils of every description should be washed in a carbolic solution after use, and subsequently boiled. The attendants ought to refrain from eating or drinking when in the patient's immediate vicinity, and should wash their hands and use a nail-brush frequently. All clothing and linen which comes in contact with the child's person should be disinfected, and washed apart from the belongings of other members of the household. A bichloride solution of 1 : 1000 may be substituted for the carbolic acid in the receptacles for linen and other articles previous to their being boiled and washed. The constant odor of carbolic acid in a private house is unpleasant, and is at times (for obvious reasons) impolitic.

¹ These statistics are included in a monograph on the "Surgical Complications and Sequels of Typhoid Fever," by W. W. Keen, M. D.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

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Synonyms.—Epidemic meningitis; Fever with cerebro-spinal meningitis; Meningeal fever; Petechial fever; Malignant purpuric fever; Spotted fever; Cold plague.

Definition.—Epidemic cerebro-spinal meningitis is a specific infectious fever (probably of microbic origin) in which the poison seems to have a special predilection for the meninges of the brain and spinal cord. It attacks the young with greater frequency than any of the fevers outside of those belonging especially to childhood, and with more severity than any of the continued fevers. The onset is abrupt (without prodromes). The prominent symptoms are chill, more or less marked; vomiting; headache; delirium, generally present in the first and second day, later stupor and coma; pains, muscular and neuralgic, in trunk and limbs; stiffness or contraction of the muscles of the neck, rarely lower down the back—all of which symptoms indicate inflammation of the meninges of brain and spinal cord. Recovery may be quite rapid, when the disease is of short duration and the nervous system is not seriously affected. In most cases, however, recovery is exceedingly slow. Death is common among children, especially in severe epidemics. The immediate causes of death are convulsions, kidney complications, exhaustion, bed-sores, and abscesses or gangrene.

If epidemic cerebro-spinal fever occurred prior to the commencement of the present century, it was not recognized as a distinct disease. It was first discovered in Geneva. In America the first reported cases occurred in Medfield, Mass., in 1806, and since that time it has occurred in frequent epidemics in different parts of North America, and in fact it is reported as an irregular epidemic visitor in all parts of the world. A sporadic form of cerebro-spinal fever is recorded yearly in the mortality statistics of all the larger cities of the United States: in studying the death-reports it must be acknowledged and remembered that some physicians call simple acute meningitis and other meningeal forms of disease, especially the continued fevers and tubercular meningitis, by the name of cerebro-spinal fever.

Etiology.—The specific cause has not been positively determined. There are physicians who have announced the discovery of a microbe similar in appearance to the pneumococcus, but it has not been satisfactorily proved that this is the specific causative germ. However, it is generally conceded that the disease is of microbic origin.

In a New York medical society meeting recently a physician stated that he had made autopsies upon 3 cases of so-called sporadic cerebro-spinal fever, and found specific germs of other diseases, all different. One had the typhoid fever germ without intestinal evidence of the disease. I am of the opinion that

when we perfect our bacteriological knowledge all these sporadic cases will be found to be due to infection of the brain and spinal cord by germs that usually affect other tissues.

Epidemic cerebro-spinal meningitis is an infectious disease, and it is questionable whether it is contagious or not. Widely-separated districts are simultaneously visited by epidemics, and over extended districts isolated individuals are attacked at the same time; so that the idea of its being transmitted by direct contact in these cases is untenable. Owing to the fact that this disease has followed epidemics of influenza, and on account of the many points of similarity in the two affections, Drs. Job Wilson and J. J. Levick have been led to suppose that there is some connection between the two diseases. It is more common in the winter and spring than in the summer months; hence the name "cold plague" has been given to it. Slight injuries, especially to the head, fatigue, exposure to cold, and mental depression are exciting causes.

Pathology.—In the early or congestive stage nothing is found in the brain and spinal cord except a congested condition of the meninges; the blood-vessels are enlarged and gorged with blood of a dark color; later, after exudation has taken place, the serous plastic exudate is found, especially upon the pia mater. In some malignant cases the exudation is found to be sero-purulent. The lungs are observed to be in a state of hypostatic congestion: where lung complications have preceded death we find evidences of croupous or catarrhal pneumonia, and not infrequently inflammation of the pleura and pericardium. Parenchymatous inflammation of the liver has been noted by some writers. Congestion and sometimes an inflammatory condition of the kidney are found. The heart is flabby, and the blood in malignant cases is frequently observed in a fluid condition. The dusky spots or mottling that are occasionally encountered in malignant cases may be found in all the internal organs as well as on the skin.

Symptoms.—The first symptom generally noticed is a chill, which may be a slight creep or a profound rigor; this usually comes on without any warning, and generally in the later part of the day; it sometimes follows fatigue or perhaps exposure to cold, and occasionally follows injuries to the head. Some cases are stricken down suddenly, as if by a blow, without any previous warning. Headache is one of the most constant symptoms; it is not always an indication of the gravity of the disease. The pain is almost always frontal, generally located between the eyes, and quite often spoken of as bitemporal; it is not infrequently located in the occipital region. It is sometimes excruciating, causing the patient to cry out and toss about; at other times it is a dull, heavy ache. It is sometimes intermittent, at others constant; it may be fixed or lancinating. The pain in the head seems to be the cause of one of the prominent facial symptoms—viz. knitting of the eyebrows. An attack may be ushered in by a convulsion, or by a sudden giddiness, causing the patient to have a staggering gait; this giddiness may only be present while the patient is sitting or standing, or may continue after assuming a recumbent position. This symptom is sometimes complained of throughout the disease.

Delirium is rarely absent; it is more apt to be noticed early in the case, extending through the stage of congestion and sometimes through the whole of the inflammatory stage; it is exceedingly variable; it may be wild excitement, terrorizing, playful, or sombre. The child may continually mutter or now and then cry out. Delirium is especially common in children, and may indicate the gravity of the disease. Coma almost invariably precedes death, and is always to be considered a grave feature; coma vigil is a serious ataxic symptom, in which the patient lies on his back, chin raised, eyelids widely separated,

apparently regarding fixedly some object above the head of his bed, and is accompanied by constant jactitation.

The headache, as before stated, often gives the appearance of great suffering, the brows being knit, especially when the patient is aroused; the cheeks are often flushed early in the disease, but not always so; later the face is frequently pale. In some rare cases the flush is not to be seen at any stage of the disease. In some patients the features are swollen and of a dull, dusky, purplish hue. Strabismus is more frequent in children than in adults.

Spinal pains are quite common, the pain being in the back of the neck, sometimes extending down to the lower end of the spine. Pressure and movement have the effect of increasing the suffering; the limbs and trunk are sometimes very painful; the pain may be of neuralgic character, radiating from centre to periphery, and may attack one set of nerves, and remain constant or change to other nerve-trunks or groups. Local muscular pains and soreness are not infrequently present.

Tonic spasms give rise to tetanoid symptoms, such as opisthotonos, pleurosthotonos, emprosthotonos: the former is the most common, the head being drawn back and the spine curved backward, so that the patient's body is sometimes supported by the occiput and heels. Forced movement increases the spasm as well as the spinal pains. In many cases these muscular spasms are a simple stiffness of muscles or groups of muscles.

Clonic spasms are frequently met with. Subsultus is one of the common symptoms, sometimes amounting to a violent agitation; more commonly it is simply a twitching, and may be the forerunner of convulsions; this symptom is sometimes present before the inflammatory changes in the nervous system are sufficiently developed to produce it; hence the reasonable supposition that it is a result of the irritation produced by the blood-poison.

Paralysis occurs as a result of a loss of nerve-power, which may be caused either by central trouble or by inflammation of the trunk of the nerve supplying the part. These paralyzes are sometimes temporary, at other times long continued or permanent. Sudden loss of hearing or sight usually comes on at the time the effusion takes place. Strabismus is especially common in children, and is often a precursor or an associate of convulsions. The conjunctivæ are quite frequently congested; at other times this symptom is absent, especially in the milder cases. In almost every case where there is kidney complication the conjunctival congestion is associated with a purulent secretion, which then becomes quite diagnostic. The pupil varies greatly; early in the disease it may be found to be dilated or contracted, but it is generally dilated. In cases with coma and convulsions it is almost invariably dilated. Photophobia is especially common in children.

The effect of the blood-poison upon the kidneys is to produce a catarrhal inflammation in these organs similar to the catarrhal troubles found in other organs.

The respiratory apparatus is involved in the disease, and some of the fatal complications are seated in the lungs. Respiration is exceedingly variable. Early in the disease it is likely to be hurried, and at times, later on, it may be exceedingly slow; it is sometimes interrupted or jerking, and the Cheyne-Stokes variety is not infrequently seen in the later stages of fatal cases. This latter is not so grave a symptom in the case of children as in adults. In some instances death occurs suddenly from paralysis of the muscles of respiration. Pleurisy, pneumonia, and bronchitis are complications which may occur at any time during the course of the disease.

In exceedingly malignant epidemics there is a dusky mottling of the skin

and the internal organs, the color being purplish; whence the name of "spotted fever" often applied to the disease. These spots (which are oval in shape) are usually from one-third to one-half an inch in their longest diameter. I have seen them on almost every tissue or organ, external and internal, of the body; after death they may be of a slate-color with a chocolate tinge, or quite black. I had an opportunity in 1864 of seeing 14 cases of epidemic cerebro-spinal fever, 4 of which died; 2 out of the 4 cases had these spots. In the Philadelphia Hospital epidemic I saw over 200 cases: the mortality was 43; of the fatal cases, 2 had these mottlings; one of them was the first case that occurred, and died after fifteen hours' illness. About sixteen years ago I was called in consultation to see two young girls near Point Breeze, Philadelphia; they both had these mottlings; one died in twenty-four, the other in thirty-six, hours. At the time only one other suspected case had occurred in the neighborhood; this also was a malignant one. The two girls had visited the abode in which this patient died.

Aside from the mottlings, there is nothing else that seems characteristic of this disease in connection with the skin. *Cutis anserina*, simple erythema, rubeoloid eruption of a bright cherry-red color in sthenic cases (darker in the adynamic), dermatitis, miliary eruptions, herpes, petechiæ, and ecchymoses, have all been noticed. Hyperæsthesia is one of the most characteristic symptoms; the skin is sore to the slightest touch, and at times the pressure of the bed-clothes is sufficient to produce great discomfort. Anæsthesia of the skin has also been observed; it may be a simple numbness, at other times a positive insensibility. In some cases the skin is found to be very hot; in others it may be quite cool; and occasionally the patient is drenched in perspiration even when the symptoms are not of a grave nature.

The temperature of cerebro-spinal fever is exceedingly varied, so that in a group of cases in the same epidemic it is quite dissimilar. The local inflammation causes changes which prevent anything like uniformity. In the explosive form, the so-called fulminant variety, it may be below normal; in all others there is more or less elevation. In some instances, early in the disease, the temperature is not very high, and in others it rises to a high elevation after the chill. When the local inflammations occur it is generally higher. In children at this stage it is usually from 100° to 101° . The diurnal variation is less than in typhus or typhoid fever. A sudden fall or rise of temperature almost invariably ushers in serious symptoms: in fatal cases it has been found at the time of death to be as high as from 107° to 110° .

The pulse in cerebro-spinal fever in children is usually quite rapid; in adults at the second and third stages of the disease it may be abnormally slow. The difference is owing to the modified nerve-influence which the disease is prone to exert.

Complications.—Among the complications observed in this disease may be mentioned pleurisy, pericarditis, endocarditis, parenchymatous degeneration of the liver and kidneys, and intestinal catarrh. "Œdema, hypostatic congestion of the lungs, bronchitis, atelectasis, and broncho-pneumonia are not uncommon lesions in cerebro-spinal meningitis" (Welch).

Sequelæ.—Parotitis; gangrene; furuncle; abscesses; muscular and mental weakness; epilepsy; impaired nerve-power, sometimes amounting to paralysis; general or special persistent emaciation; and, in children, effusion following the inflammation of the membranes of the brain sometimes results in chronic hydrocephalus. Dr. Chas. K. Mills, in a paper read before the Philadelphia Neurological Society in March, 1888, called attention to the occurrence of multiple neuritis as a complication of this disease, and also suggested that multiple neuritis might be the only result of the same infection that causes the meningitis.

Diagnosis.—In the earlier stages, especially in children, it may be mistaken for scarlet fever. This is true where there is a general erythema or dermatitis. The existence of the epidemic influence of either disease or the presence or absence of severe throat symptoms will greatly assist in the diagnosis. The redness of the skin coming on in epidemic cerebro-spinal fever generally appears later than that of scarlet fever, in which it usually happens in the first twenty-four hours. The eruption is quite transitory, and is not, as a rule, followed by desquamation or itching.

The abrupt onset and the greater activity of the symptoms, the absence of tubercular manifestations elsewhere, the rarity of eruptions and extreme muscular contractions, the slow regular course, and the higher temperature would distinguish epidemic cerebro-spinal fever from tubercular meningitis. The absence of exciting causes, the extremely faint muscular spasms, and the sensitiveness of the skin, all help in distinguishing it from simple or secondary meningitis.

The muscular spasms and general and muscular pains usually distinguish this disease from ordinary cases of pneumonia, typhus, and typhoid fever; but in the meningeal forms of these diseases it is extremely difficult to make a diagnosis, though the sudden onset with meningeal symptoms will greatly assist. The earlier symptoms should be studied to find out whether there were evidences of pneumonia or any other previous disease. Abdominal symptoms occurring early might suggest typhoid fever. The eruption of typhus is the distinguishing mark in that affection. Rigidity of the muscles, present in cerebro-spinal fever, is absent in the preceding diseases. I have known mistaken diagnoses to be made in cases of small-pox in the earlier stages.

Prognosis.—This is always grave in children, more so than in adults. When we take into consideration the extreme susceptibility of the nervous system of a child, we can readily see how dangerous this disease is during the earlier years of life. Prognosis in adults is a difficult task, for in simple cases sudden grave complications sometimes present themselves later in the disease, and, on the other hand, a case with the severest early symptoms may be followed by speedy convalescence. It is a disease in which it is impossible to estimate the complications which may arise.

Unfavorable signs are profound coma; low typhoid symptoms; uræmia; great blood dyscrasia, shown by marked ecchymosis; continued convulsions and prolonged high fever. Protracted cases are likely to be followed by fatal exhaustion.

Treatment.—The types and lesions of the disease are so various that the details of the treatment are exceedingly difficult to formulate to meet all cases.

The prophylactic treatment consists in careful attention to sanitation, as the disease is invited by uncleanness of person or surroundings; the same is true of over-crowding. Exposure to heat or cold, and fatigue, either bodily or mental, are favorable to the onset of the disease. Children in a locality where the affection is prevalent should be furnished with fresh, nourishing, and easily-digested food; they should be isolated from the sick, and should have plenty of sleep and pure air. Clothing from about the sick should be destroyed or carefully disinfected. The weak, old, and nervous should be removed from infected localities.

Almost every remedy in the medical category has been tried to abort this disease: bloodletting has had its votaries, and others have highly extolled the virtue of mercurials in the earlier stages; emetics, again, have been recommended, but all have largely been abandoned. The plan pursued by most recent authorities is to treat the disease symptomatically.

In the first stage we have a congested condition of the meninges of the brain and spinal cord: the indication is to aid in the reduction of the quantity of the blood in the meningeal blood-vessels; first, for the purpose of relieving the symptoms, and, secondly, to reduce the inflammation and modify the inflammatory products. One of the difficulties of administering medicine by the mouth is the common symptom of vomiting, which is sometimes very persistent. Venesection should not be practised in children. Some of the German writers use early local bloodletting by wet cups and leeches. Dry cups to draw blood from the internal congested vessels without removing it from the body are of great value. The external application of cold to the head by ice, ice-water cloths, cold-water cloths, is useful, and some have used hot baths to the body, hoping to draw blood from the centre to the periphery. Hot mustard foot-baths can be used with advantage to relieve the pain in the head and back. If the stomach should bear it, potassium bromide and ergot may be administered; if not, the former may be given by enema, the latter hypodermatically, for the purpose of favorably influencing the capillary congestion. For the pain in the muscles the antipyretics have been used; phenacetin is probably the safest and best of all. It should be used in small, frequently-repeated doses, and its use should be discontinued if the patient becomes weak or exhausted. A mustard plaster, one part mustard to three of flour, placed over the spine, often relieves the pain in that location, and counter-irritation to the nape of the neck diminishes the pain in the head and relieves the delirium. Care should be taken not to raise a blister, which would seriously complicate the case. Liniments over the same region—turpentine or chloroform—may be used for similar purpose. Belladonna seems to afford relief to the neuralgic pains and muscular spasms. Dr. J. M. DaCosta highly lauds the use of hyoscyne hydrobromate for the muscular spasms in this disease. For insomnia early in a case chloral may be cautiously used in conjunction with potassium bromide. Chloral sometimes causes cerebral excitement, and when this occurs it should be discontinued. Opium has always been used with the happiest results. It has been recorded that in some cases large doses of opium are tolerated. The salicylates and gelsemium will allay the pains in the trunk and limbs, but will not relieve the pain in the head. A dark, quiet room should be selected for the patient in any stage; this is of great importance where there is cerebral excitement.

In the second stage the exudate is thrown out; it may be serous, plastic, or even sero-purulent; the blood-vessels are dilated and engorged. Absorptive remedies are now to be used. Potassium iodide to produce absorption of the exudate, and oil of turpentine internally have been used late in this stage for the same purpose, with seeming good results. Arsenic and iron are of great use during convalescence to improve the blood. Stimulants, especially for children, should be used with great caution, as an excess will irritate the brain and excite the circulation in either the first or second stage. Hypophosphites, especially with strychnine, are beneficial during convalescence. Cod-liver oil when digested often produces the happiest results. In the later stages of convalescence massage is of great importance to stimulate the circulation in the muscles and nerves. Electricity is indicated for paralysis or weakness of the nerve-trunks. For the same purpose alternate hot and cold affusions to the weakened parts, and exercise, carefully regulated as to time and amount, greatly assist in strengthening the muscles and nerves.

EPIDEMIC INFLUENZA.

By CHAS. WARRINGTON EARLE, M. D.,

CHICAGO.

INFLUENZA is a general infectious disease producing catarrhal difficulties of either the respiratory or gastro-intestinal tract, or painful symptoms referable to the nervous system. In addition to the symptoms thus indicated, it is attended with prostration out of proportion to the apparent involvement of the organs named, and is liable to be followed by sequelæ which affect profoundly the further usefulness and comfort of the unfortunate victim. This disease has been recognized and described in our country for two hundred and fifty years, the first epidemic occurring about 1647. Other epidemics have taken place from time to time, and have been referred to by writers under different names; but the disease, as it affects us particularly, and its history, as we understand it at the present moment, have come to us in the three consecutive epidemics of 1890, 1891, and 1892. At the time of writing (January, 1893) only a few sporadic cases have taken place during this year, and they have not been severe. We cannot yet speak of an epidemic of 1893. During the period referred to, great attention has been given to the study of the disease by our profession, and, in certain instances, by governmental authorities.

Etiology.—It has not been believed until recently that the causes of this disease are really known. Certain hypothetical causes have been advanced, such as air, contagion, local conditions, general influences, etc. But during the last three or four years very close investigations in regard to its etiology have been made. The reports of the British medical government clearly show that the spread of the disease depends upon human intercourse, and that it spreads no faster than human beings, parcels, or letters can travel.

Bacteriological investigations have been carried on with great accuracy during this time. Filatow wrote fully concerning the history and symptoms of the disease under consideration, and Seifert investigated the bacteriological history three or four years ago; but particular investigations have been carried on during the past year in the Berlin Institute by Drs. Pfeiffer, Kitasato, and Canon; and Sternberg remarks that there is good reason to believe that the bacillus discovered by these investigations is the specific cause of the disease. The following résumé from Dr. Sissley of London gives much regarding the etiology of the scourge under discussion:

(1) The first case of influenza in a town is generally a patient who has come from an isolated place.

(2) Isolated cases precede the epidemic.

(3) Influenza extends along the lines of human intercourse.

(4) Isolated persons, such as prisoners and inmates of asylums and convents, often escape the disease.

(5) The number of those affected in an epidemic increases till a maximum is reached, and then declines, as in the case of other contagious diseases.

There is no doubt that nursing children three or four months of age feel the influence of la grippe. Dr. Townsend of Boston has placed on record a case where the mother had an attack of influenza about the time of her confinement, and the child in a few hours after birth began to sneeze and had all the symptoms of this infection; and an English observer records the case of an infant who died on the third day of its life from this disease. It is somewhat difficult to diagnosticate influenza in very young infants, but it is fair to suppose that, when the infection is present in the house and parents and nurses are under its influence, if infants present unusual symptoms of fever, exhaustion, and the involvement of one of the three systems which are usually selected by this infection, the disease is due to the poison of influenza.

The exact point at which the infection may gain entrance to the system has probably not been ascertained. That it may enter through either the alimentary canal or the lungs there is no doubt, and in all probability these are usually the points of entrance. One observer believes that the conjunctiva is in many instances the structure through which the poison attacks the system.

Influenza and Diphtheria.—The marked similarity between the remote effects of the poisons of diphtheria and influenza is very great, and it is quite possible that the pathological findings in influenza may be quite as numerous and significant as we already know they are in diphtheria. We possibly do not know the exact cause of influenza, but we are certainly warranted in assuming that there is a most profound toxic effect in influenza as well as in diphtheria. The depression is profound, the recovery slow and tedious, and the involvement of the nervous system in both diseases is extremely significant. The action of these two poisons upon the heart is somewhat similar. Every practitioner of experience has noticed the slowness of the pulse and its irregularity, and in some instances death has occurred in such an unexpected manner that we could attribute it to nothing less than degeneration of the heart-muscle.

Pathology.—There are but few special post-mortem findings known to this disease which are of value to us as relating to children. Nearly every study has been based upon examinations made in adults, and the records of autopsies made solely and particularly to find the results of influenza on the tissues of the young are extremely meagre. Ashby and Wright state that “at the post-mortem no grave lesion is found, but there is usually venous congestion and marked injection of the venous capillaries;” and Vargas of Barcelona, whose opportunities for seeing many cases profoundly sick with influenza have certainly been very great, after remarking that rapid deaths are usually due to severe attacks affecting the nervous system, says that while we cannot state that there is an apoplectic form, in some cases the post-mortem revealed the venous plexus congested, and also cerebral hæmorrhages. The same author also asserts that in cases where the gastro-intestinal symptoms predominated there was tumefaction of Peyer’s glands and of the solitary follicles.

In 115 references to influenza found in the *British Medical Journal* of 1891 and 1892, not one speaks particularly of the pathology as it is found in children. And in the works of Filatow and Uffelmann, both written in 1892, absolutely nothing is said regarding this part of our subject. The special effects of the poison of influenza upon the tissues of the young have yet to be described.

Incubation.—This may be only a few days, possibly only a few hours, or, on the contrary, the influence of the poison may be felt for weeks before the active development of the disease. Others who have studied the disease believe that two or three days is the usual time of incubation.

Clinical History.—The disease affects more particularly one of three groups of organs: First, the respiratory and circulatory apparatus; second, the gastro-intestinal canal; third, the nervous system.

Sometimes the infection localizes itself in the respiratory tract, spending its energy there, and the patient will pass through a severe catarrhal bronchitis or a pneumonia with such general prostration as to endanger his life; or the disease manifests itself as a catarrhal inflammation of the stomach and bowels, with a tendency to collapse on account of the extreme weakness which is induced; or, closely following the severe headache, which indicates that the nervous system is the first to be attacked, have come threatened convulsions and meningitis. We have these organs affected singly, or in some cases a complication involving almost all of them, such as a bronchitis with gastro-intestinal disturbance, or a gastro-intestinal disturbance with great nervous prostration.

The invasion is rapid, and the disease is frequently ushered in with a chill followed by delirium and rapidity of pulse. The face in many cases is red from the commencement of the disease, and there is earache, vomiting, and an increase in temperature. The fever is not high in the majority of cases, but occasionally an unusually high temperature is noticed. In a majority of cases, at some time during the disease, the temperature is subnormal, varying from one-half to two degrees below the standard of health. This condition of temperature is undoubtedly a result of the action of the poison upon the general nutrition, the imperfect action of the lungs which is present in many cases, and the general depression of the vital forces. There is also loss of weight. This has been particularly brought out by Hansen of Copenhagen, who concludes that, while in some cases there is simply a standstill, in many there is an absolute diminution in normal weight. It is fair to conclude that this evidence of waste—in other words, work—represents the conflict between the poison of influenza and its subjects. In some cases this diminution of weight is noticed when there are no other signs of the disease present. And finally there is a very pronounced general weakness never before experienced by the patient, and in no one organ or system of organs is it more noticeable than in the circulatory apparatus. The pulse is usually accelerated, sometimes very rapid, and the heart, in many instances, never regains its strength and vigor.

Special Features.—*Respiratory Symptoms.*—A catarrh of the respiratory organs takes place with great frequency, and in its various phases extends to every part of this system. Sometimes the upper breathing apparatus is attacked first, and the disease rapidly spreads and involves the rest. The eyes are usually red and suffused, and in many cases not only is the middle ear involved, but disease of this organ remains as a sequel for a long time. A general catarrhal bronchitis is frequently present, and in some instances pneumonia with all its characteristic symptoms. There is in many cases, early in the disease, an apparent localization of the infection in one or both of the lungs, threatening a pneumonia, but this usually clears up in a very short time, and the disease becomes diffused throughout both lungs. Very often there may be only a severe and perplexing cough, without any physical signs. Respiration is sometimes slow, and in a few cases breathing for a few seconds has absolutely stopped. These peculiar paroxysms have been repeated several times during the day, and in a few instances life has been preserved during these attacks only by artificial respiration. Thoracic pains are sometimes intense, and call for the external application of anodynes.

Circulatory Symptoms.—There is usually from the first a rapidity and weakness of the heart, and syncopal attacks occur in many cases. Depression in the action of this organ and failure in its supply of nerve-force seem entirely

out of proportion to all other symptoms. While in many cases the temperature and pulse seem fair, there is an unusual muscular weakness and a tendency to syncope. I have not noticed organic heart disease, but cyanosis has been present in a few cases, and in many instances palpitation and short breathing are not only noticed during the active history of the disease, but also interminably follow its unfortunate victim.

Gastro-intestinal Symptoms.—The tongue is frequently flabby and coated, and shows indentations of the teeth, indicating malnutrition. The appetite is often entirely absent, and persistent vomiting takes place in many cases. Herpes labialis is sometimes noticed, as also sordes. Diarrhœa to such an extent as to become exhausting is frequent; constipation is sometimes present. In some cases the diarrhœa and vomiting are so frequent and persistent, and the child becomes so rapidly collapsed, that if the case occurred in the summer a diagnosis of cholera infantum would undoubtedly be suggested. As the result of this great withdrawal of fluids from the body, the eyes and fontanelles are greatly depressed, and the child becomes restless and rapidly goes into collapse.

Nervous Symptoms.—Extreme irritability and fretfulness are found in the majority of childish patients. Headache and joint and muscular pains are frequent and sometimes intolerable. In many cases there are noticed an indifference and a hebetude which closely simulate a typhoid condition. Convulsions take place in a small percentage of children, and congestion of the brain with drowsiness may be noticed. In one case which came under my observation the child did not close its eyes for four nights. It was not unconscious, but indifferent, and wanted to be left alone. In a few cases meningitis will seem imminent, and the diagnosis will sometimes necessarily be held in abeyance. In some children afflicted with influenza there is developed an obstinacy which is truly remarkable; they sometimes resist the slightest touch, and refuse all examination on the part of the physician. This peculiarity is regarded by some observers as of diagnostic importance in differentiating from typhoid fever.

Temperature.—In addition to what I have already said, I have noticed that the fever may be very high and yet recovery take place. On the other hand, a temperature of 101° to 102.5° F. may persist for a period of two or three months. In these cases I have suspected and have repeatedly examined for evidence of tuberculosis, and have not found it, the patient finally making a good recovery after this long period of sickness. In general, we may make the statement that the temperature is more irregular in influenza than in any other disease.

Complications and Sequelæ.—These are numerous and varied, and attack nearly every function and organ of the body. Glandular enlargements are frequent. Inflammation of the parotid gland may take place. Abscess of the antrum and inflammation of the connective tissue of the neck have been noticed. Tuberculosis and tubercular meningitis may follow in a few cases. Conjunctivitis may remain, and catarrhal inflammation of the middle ear, resulting often in perforation and profuse discharge, will be noticed. At times this involvement of the middle ear, while always a serious complication, may even threaten the life of the patient. Diseases of the skin are sometimes noticed, such as erythema, herpes, and urticaria.

Among the more general diseases that have been observed are rheumatism, chorea, nephritis, and periostitis. Children having a tendency to rickets have been known to develop the disease after having had an attack of influenza. Among the complications which I have noticed, and which I have not seen

recorded, is purpura. Of this I have seen four cases, all in young people, and attended with extreme weakness and with evidence of more or less blood-change.

As is not unusual in adults, acute mania has been observed to follow the disease occasionally in children, but generally ends in complete recovery. Dr. Julius Althus, in an extensive article on mental affections after influenza, gives cases illustrating neurasthenia, hypochondriasis, melancholia, delirium from inanition, homicidal tendencies, and general paralysis. He believes that the psychoses observed after epidemic influenza are far greater than those after any other infectious disease.

Diagnosis.—From the rapidity with which it seizes the patient, influenza might be mistaken for sunstroke, an acute poisoning, or malignant malaria. It can be confounded with all diseases of the respiratory apparatus, with typhoid fever, and with meningitis.

From a simple catarrh, influenza will be distinguished by the fact that it is epidemic, and that there is greater prostration, which continues for a longer period of time, than in the first-named disease. The temperature is also higher, and there is a tendency to catarrhal difficulties—at first local, but rapidly spreading to other portions of the body. A mild catarrh, with severe neuralgia and with unusual pain in the limbs, should be diagnosticated as influenza if this disease be prevalent. The same may be said in regard to an irritable stomach, with diarrhoea and an unusual prostration. This in a time of epidemic should certainly be classed as influenza. From pneumonia and bronchitis, simple or capillary, we differentiate influenza by the absence of the usual physical signs, although at the commencement of the grippe in many cases there will be symptoms of pneumonia, and it seems as if localization had indeed taken place; but frequently in a few hours this becomes diffused, and a general bronchitis with the excruciating pain and prostration belonging to influenza is detected.

From typhoid fever influenza is differentiated by the fact that no rose-spots appear and no enlarged spleen is found, and the catarrhal condition, more particularly in the respiratory tract, predominates over all other symptoms. If diarrhoea exists in influenza, it will be noticed that a cough and a catarrhal state of the air-passages has preceded its development. The fever in influenza is irregular; in typhoid it is so regular and constant that it almost makes its own diagnosis. It is not usual to notice the apathetic facial expression that we have in typhoid. The face, however, is usually flushed in influenza—more frequently pale in the continued fever. There are no rose-spots in influenza, no tenderness and gurgling in the right inguinal region.

From meningitis influenza can usually be diagnosticated by careful observation of the eye and by the want of the rigidity of the muscles which we find in meningitis. The disease of the brain usually develops rapidly, and if death does not take place it disappears quickly. I must, however, say that the differentiation of meningitis from certain forms of la grippe is attended with great trouble, and a diagnosis must in some cases be withheld. When the fever persists after all other symptoms of influenza have subsided, and there is a cough with gradual emaciation, the closest care must be taken that a tubercular disease does not come in. Particular attention should be given to nutrition, and every means should be taken to diagnosticate the disease early.

Prognosis and Mortality.—In this connection an interesting topic might be discussed as to whether one attack of influenza protects from subsequent attacks. I do not think that this question at present can be fully answered, but the general statement can be made that many families particularly afflicted

in 1889 did not develop the disease in 1890 or 1891. There are those who are immune from the disease, and others in whom it has developed three consecutive years.

The mortality is different in different epidemics, and the character of the epidemic must be considered, as in all other infectious and contagious diseases. In some epidemics children are particularly liable to contract the disease, while in others adults seem to be selected. And again in a more general epidemic it has been noticed, as I can personally attest, that children often are not attacked until the disease has prevailed for some time. When the attack is moderately severe, I regard it a dangerous malady for a child, particularly if he has anæmia or any vicious constitutional tendencies. Death has taken place in twenty-four hours. It may come from almost every complication, but, in the main, exhaustion and bad nutrition bring about the fatal result. Death may come with such rapidity that in summer insolation is suggested, and at other times malignant malaria. In the fulminant variety with rapid death, the severe symptoms will be referable to the nervous system, while throughout the entire history of other cases the poison selects the respiratory or gastro-intestinal tract, and death comes as it does in those diseases when not complicated with influenza. But it must be remembered that there is always a tendency to collapse and a prostration out of proportion to other symptoms.

The length of time consumed in convalescence from this disease is wonderful. The pains and general weakness do not disappear for weeks; and I may add that many of the sequelæ remain for years, and not only produce suffering, but shorten the life of the individual.

Treatment.—I have no particular remedy or combination of remedies to suggest. I think, however, that care should be taken to prevent the contagious element from spreading and gaining a hold on the community, and, in view of the great mortality and the immense money loss which this disease causes, it appears to me that the time will come when it will be regarded as the duty of all municipal authorities to assume such control of the disease as science suggests.

Let the people understand that it is a contagious disease, and instruct them how to prevent its spreading by contact. All handkerchiefs and cloths used by the patient must be immersed in some antiseptic fluid, and all cuspidors and articles of furniture which come in contact with the germs of the disease should be carefully disinfected.

A generous diet must be insisted upon, some stimulation, and a conservation of all the strength of the patient observed from the outstart.

For the general pain which pervades the entire system, which sometimes is the first and most prominent symptom, nothing has given me such good results as phenacetin and salicylate of sodium. The catarrh of the respiratory tract which speedily prostrates young children should be early treated with stimulants, including the ammonia preparations and the ordinary expectorants. The gastro-intestinal catarrh must not be neglected, but should receive attention from the first. It is a clinical fact, which must have been observed by many, that in some of the neglected cases there is just as profound and general collapse from the copious diarrhoeal discharges and vomiting, which we sometimes see in this form of the disease, as from those which take place in severe cases of cholera infantum. They should, then, have attention from the very first. For the extreme fatigue and depression not only alcoholic stimulants, but the effervescing waters with quinine, should be administered. If the stomach is particularly irritable, let the quinine be administered by inunction or by the rectum. Children take eagerly and with good results whipped egg-albumin

with sterilized water and a little stimulant and sugar. Champagne is excellent for the depression which is so evident among these little people. When there is great prostration following the involvement of any of the three systems we have mentioned, the carbonate of ammonium, camphor, and musk, fortified by the conjoint use of digitalis and nux vomica, are indicated.

When the patient begins to pass out from the more painful and acute manifestations of the disease, in addition to a generous diet a tonic composed of the compound syrup of hypophosphites, extract of malt, and pepsin cordial, equal parts, with a very small amount of elixir of bark, iron, and strychnine, acts efficiently.

ERYSIPELAS.

By FREDERICK A. PACKARD, M. D.,

PHILADELPHIA.

ERYSIPELAS is an acute, specific, contagious, inflammatory disease of skin and mucous membranes, accompanied by marked general symptoms, and characterized by peculiar local lesions at the seat of inoculation, by its tendency to spread, and by the presence in the affected area of a micrococcus that is capable of reproducing the disease in other individuals.

The word "erysipelas" is probably derived from *ἐρυθρός*, *red*, and *πέλλα*, *skin*. Numerous qualifying words have been used to signify the point of involvement, the course of the disease, the appearances presented by the local lesion, the age at which the disease occurs, etc. The terms "traumatic" and "idiopathic" have been used to distinguish cases wherein there is or is not an antecedent obvious wound of the skin at the seat of the local lesion. No qualifying words should be used as implying an essential difference in the process, as it is a disease *sui generis*, no matter under what circumstances it may occur.

History.—Erysipelas has been known from the time of Hippocrates, but the descriptions of the disease given by most writers prior to those of the last century show that many diverse diseases were included under this name. When humoral pathology occupied men's attention, this, in common with many other maladies, was supposed to be the outward expression of morbid humors in the body. At a later date it was looked upon as a simple dermatitis; still later, as a simple lymphangitis. The contagiousness of the disease was pointed out by Lorry in 1777. A microbic origin was first suspected by Martin in 1865. The question of priority in demonstrating this origin is still a matter of dispute. Between 1868 and 1870, Nepveu and Hueter described the occurrence of microscopic organisms in connection with the disease. It need only be stated here that the description given by Nepveu corresponds more closely than does that of Hueter to the micro-organism now established as the cause of the disease. Since 1870 many observers have studied the disease from a bacteriological aspect, but it is especially to Fehleisen that we owe our present knowledge of the life-history and etiological rôle of the micrococcus described by him in 1882.

Etiology.—The disease is limited in its occurrence to no part of the civilized world, but its favorite habitat is the temperate zone. It but rarely occurs in the tropics, being less rare in regions far removed from the equator. In Greenland, for example, occasional widespread epidemics have occurred.

The predisposing effect of season can be readily seen by the accompanying chart (Fig. 1). It will there be found that by far the greater number of fatal cases in Philadelphia occur during the latter part of the first and the early portion of the second quarter of the year; that is, during the early spring months. Allen analyzed 566 cases applying for treatment, and obtained practically the same result.

It appears to be most prevalent among the poorer classes. This may be due to several causes—the greater liability to injury, frequency of chronic

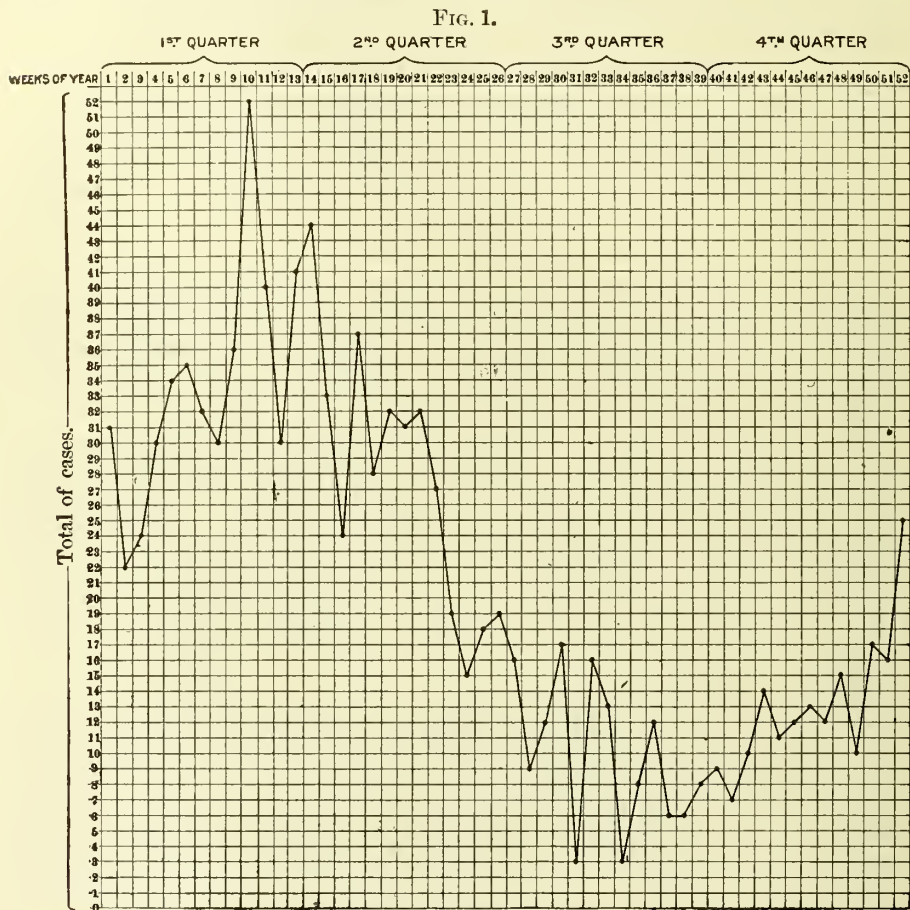


Chart showing the Number of Fatal cases of Erysipelas in Philadelphia occurring in different seasons from 1874 to 1891.

superficial inflammatory troubles, lack of cleanliness, want of ordinary sanitary precautions, and neglect of proper isolation amongst those attacked.

The question of age as a predisposing factor is difficult to determine, as only fatal cases appear in the reports of boards of health. Of 12,556 fatal cases of the disease in England between the years 1862 and 1868, there occurred under one year of age 31 per cent.; under five years, 5.9 per cent.; under fifteen years, 2.9 per cent.; under twenty-five years, 4.2 per cent.; under forty-five years, 12.4 per cent.; under sixty-five years, 20.9 per cent.; above eighty-five years, 1.4 per cent. In Philadelphia, during the period between 1874 and 1891, there occurred 1253 deaths from erysipelas. Of these, 380 were in children under one year of age, 35 between one and two, 23 between two and five, 25 between five and ten, 6 between ten and fifteen, the remaining 784 cases occurring in those past the latter age. All that can be said, therefore, is that no age is exempt. The large number of fatal cases occurring in the first year of life may be due to the almost uniform fatality of the disease during the early part of that period, and cannot be taken as an index of the actual number of cases occurring in infants.

What part filth and defective drainage may play in its production has not been definitely settled. In the older hospitals of Europe frequent epidemics have occurred; but it is not alone in these that erysipelas appears, new and apparently sanitary institutions being also the scene of its occurrence. A well-known and oft-quoted instance of the effect of polluted air is that which occurred in the Middlesex Hospital, where a defective drain was on two occasions the apparent cause of an outbreak of the disease, starting in the bed nearest to its position in the wall. It is said to be frequent in the immediate neighborhood of badly-kept stables.

The most important etiological factor is contagion. The contagious principle has but a limited area of influence, as is shown by some of the histories of local epidemics within hospital wards, wherein patients upon one side of a ward have been affected *seriatim* on both the right and left of the individual first attacked. Those in attendance upon a case are apt to contract the disease. One attack seems rather to predispose to, than to protect against, a recurrence, due probably to the fact that some breach of the surface produced by a chronic affection admits the poison.

The contagious principle is the *streptococcus erysipelatis*. Although previous investigators had discovered micrococci in the local lesion, the most careful and conclusive work upon the subject was performed by Fehleisen, hence the micro-organism is frequently spoken of as the streptococcus of Fehleisen. By him it was found in the lymphatic vessels and spaces of the skin and subcutaneous cellular tissue, and in the superficial layers of the corium. It occurs as a single cell or in the form of diplococci or chains of various length. The individual cell measures about 0.3μ in diameter. It is readily cultivated upon gelatin and blood-serum, where the colonies form as dull-white, round points, closely margined or fusing at points of contact. It grows well at the temperature of the human body, is facultatively aërobic, and develops well *in vacuo*. Not only has the inoculation of pure cultures been successfully practised upon animals, but the disease has been inoculated upon human beings as a therapeutic measure.

In order that the parasite may gain access to the lymph-spaces, it is essential that some breach of the surface should exist. This means of entry may be supplied by some wound accidentally received or purposely inflicted, by the unhealed navel of the new-born, scarifications made for purposes of vaccination, the local lesion of vaccinia, the ulcers of varicella, solutions of continuity produced by eczema, intertrigo, ecthyma, or pemphigus, or by ulcers resulting from chronic inflammation of the mucous membranes of the mouth or upper air-passages. It is owing to the frequency of lesions at the points of union of skin and mucous membrane that the local manifestations frequently begin at those situations.

Pathological Anatomy.—After death the body-heat is maintained for a long time, and, according to Eulenburg, there is a post-mortem rise of temperature to a point $.9^{\circ}$ C. (1.5° F.) above that observed before death.

At the seat of the local lesion the vivid color gives place to a mere yellowish discoloration, and much of the swelling observed during life disappears. When the skin is incised there exudes a varying quantity of more or less discolored serum. The skin and subcutaneous tissue are somewhat thickened and cannot be readily separated. Microscopical examination of the affected skin shows that beyond the peripheral margin there are numbers of micrococci in the lymphatic vessels. As sections are made from without inward, the greatest histological changes are seen at the visible margin of the patch, where there are much serous infiltration separating the cells, and infiltration by round

and wandering cells, many micrococci being contained in the latter. From this point the alterations progressively diminish as the part earliest attacked is reached, until complete *restitutio ad integrum* is found to have occurred. The hair-shafts are unaltered, but there is serous and cellular infiltration of the root-sheath, and micrococci may be found in the space between the latter and the root. In lately-developed vesicles upon the surface no micro-organisms are to be found, but in those of longer existence various forms abound. In phlegmonous erysipelas there is an admixture of the staphylococcus pyogenes with the streptococcus erysipelatis.

The mucous membranes that are affected show the same appearance as does the skin, save for the normal structural differences in the tissue. Attacking the larynx, the disease produces marked swelling in the parts around the glottis. Edema of the rima glottidis may be present. The trachea and bronchi may be of a brilliant red color, with paler areas corresponding to the cartilaginous rings. Three forms of pulmonary lesion may be found: (1) an accidental croupous pneumonia, with the ordinary appearances of that lesion; (2) intense congestion, either general or limited to diseased branches of the bronchial tree, with scattered areas of red or gray hepatization within the congested area; (3) an acute infective interstitial pneumonia from bacterial embolism, with subsequent dissemination of micrococci in the interlobular connective tissue. In cases where the disease has spread from the air-passages the alveoli contain large numbers of leucocytes and many micrococci, instead of the fibrin and epithelial cells seen in croupous and catarrhal pneumonia.

Inflammation of the pleura may be found from extension of the disease through the chest-wall or as secondary to subpleural pulmonary lesions. The pleural cavity may contain serous or purulent exudate. The streptococcus has been found in pleural exudate. Suppurative anterior mediastinitis has been observed. Pericarditis is rarely seen, but endocarditis, affecting chiefly the free borders or the whole of a leaflet of the valves of the left side, is occasionally present. Granular degeneration of the myocardium also occurs, due doubtless to the elevation of temperature. The endothelium of the blood-vessels has been found to be swollen, granular, and with indistinct nuclei. Tutschek reports a case of thrombosis of the abdominal aorta. The streptococcus has been found in the blood of the skin, subcutaneous adipose tissue, and in the capillaries of the lungs, liver, spleen, and kidneys.

The stomach may exhibit marked engorgement of its vessels, the intestinal tract patchy redness. Multiple minute duodenal ulcers have been seen. In the large intestine the typical erysipelatous local lesions may be found in cases where the disease has spread from the perineum through the anus to the rectal mucous membrane.

The liver may be large and congested in rapidly fatal cases; in those of longer duration it is more often pale, soft, and the seat of fatty degeneration. Many observers have found the streptococcus within the organ.

By most authors the spleen is said to be increased in volume, as would be expected from the frequency of its enlargement during life in non-fatal cases; but Denucé found it small, soft, and hyperæmic.

Peritonitis is comparatively rarely found, most instances of its occurrence being in the new-born, where the abdominal wall has been the seat of the primary process.

In spite of the prominence of cerebral symptoms during life, there are but seldom found any marked structural alterations within the cranium. The membranes may be anæmic or their vessels intensely engorged with blood. Actual meningitis is rarely seen. An instance is reported by Osler of menin-

gitis and thrombosis of the lateral sinus in a fatal case of facial erysipelas wherein the process could be traced along the trunk of the fifth cranial nerve.

From the frequent presence of albuminuria it is to be expected that in fatal cases the kidneys would show structural alterations. In five cases examined by Denucé these organs showed nephritis in degrees varying with the duration of the case. Langer has reported a fatal case of erysipelas of the scalp occurring in a seven-weeks-old boy, and complicated by hæmoglobinuria, wherein the kidneys showed infarcts and miliary abscesses. In the articular inflammatory exudate that sometimes occurs Schüller found the streptococcus.

Symptoms.—In spite of the fact that in six cases purposely inoculated by Fehleisen the initial chill occurred in from fifteen to ninety-one hours, the incubation for cases accidentally inoculated may be reckoned as requiring a period of from three to seven days.

The onset may be sudden, the first symptom being a chill with rigor. In other cases feelings of languor and vague discomfort in the part that later becomes the seat of the local lesion may precede the occurrence of chill. In young children the occurrence of an initial convulsion is not infrequent. The attack may begin with severe inflammation of the upper air-passages or throat, the skin lesion not appearing for twenty-four or thirty-six hours after the first signs of illness. The temperature rises rapidly to 102°, 103°, or even 105° F. The affected area soon becomes the seat of burning, smarting pain. The local appearances at this time may merely amount to slight redness and glossiness. In a short time there is slight elevation of this reddened area above the surrounding healthy surface, the color deepens in shade, and there are pitting and pain upon pressure. The color is readily dispelled by pressure, but quickly returns upon the withdrawal of the finger. The pain becomes more intense, and there is a sensation of stinging and stretching in the affected part. The tongue is coated, there is anorexia, thirst may be marked, varying degrees of cephalalgia are present, while nausea is a frequent source of complaint. Vomiting is not frequent in cases of ordinary severity. At this stage the pulse is usually full, bounding, and rapid. Upon the second day the temperature-chart shows a slight morning remission. The redness and swelling extend from the original site to cover a larger area; the eyes may be invisible from swelling of the lids, the ears swollen and distorted, and the lips thickened. Cephalalgia becomes intense, especially if the scalp be invaded; insomnia and delirium frequently appear. Albuminuria, with a copious deposit of amorphous urates, will usually be found after the first few days. On the second or third day the local appearances of the part first attacked reach their highest degree of development. Thereafter the redness and swelling of that part subside. Meanwhile the local process may have steadily advanced from the point of its original appearance until large areas of skin are involved. When extension ceases the temperature rapidly falls, the pulse becomes less bounding and its frequency diminishes, pain lessens, the associated symptoms rapidly subside, and the patient enters upon convalescence. During convalescence the affected skin has a faint yellowish discoloration and is the seat of desquamation, the epidermis separating in branny scales or in large flakes, and in cases where the scalp has been invaded the hair falls. Albuminuria may persist in lessening degree for several days after the cessation of other symptoms.

Important variations from this ordinary type occur and require separate consideration.

Erysipelas of the new-born begins either at the navel or at a point nearer to the symphysis pubis. Thence extension rapidly occurs until the skin of the whole abdomen, that of the extremities, or even larger portions of surface, may

be involved. The infant exhibits extreme restlessness and has high fever, may vomit frequently, and soon passes into an asthenic condition that speedily ends in death. In other cases the process extends along the still patulous umbilical vein, reaches the liver, and may lead to fatal peritonitis. After the early days of infancy are passed the disease shows the same characters in children as in adults.

Where the mouth, tonsils, pharynx, or nares are primarily attacked, the local appearances are those of an intense inflammation of the part affected, but swelling is more marked than usually occurs with ordinary inflammation, and the tendency to spread to adjacent structures and the skin is a peculiarity of great diagnostic importance. From the nares it may extend to the lachrymal duct and attack the skin near the internal canthus. From the upper air-passages the process may extend to the bronchi or to the lungs, producing the symptoms and physical signs of an intense bronchitis or pneumonia. In the primarily laryngeal form hoarseness begins early, and may be rapidly followed by symptoms of suffocation due to the intense swelling of the mucous membrane.

The eruption exhibits certain peculiarities worthy of further study. Extension usually takes place most rapidly in one direction, but not in an even line, as flame-like tongues of redness frequently jut out in advance. The area of redness and swelling is bounded by an abrupt fall to the level of the healthy surface. Extension from the face usually occurs upward, reaching the hairy scalp or even passing backward to the nape of the neck or to the trunk. From the trunk it may spread to the extremities or head, and *vice versa*. One striking peculiarity of the eruption is its liability to terminate at natural boundaries—the borders of the hairy scalp, the various folds of the face, the groin. Where the underlying bone is close to the surface the eruption is frequently absent; thus the chin may be spared, while the rest of the face is much swollen. Conversely, where the skin is but loosely attached to underlying structures—as in the scrotum, labia majora, and eyelids—swelling is very marked, and gangrene may occur from interference with the circulation. Besides redness and swelling, other appearances are usually present in the affected area of skin. Vesicles, or even bullæ with clear or muddy contents, are apt to form. Pustules are rarely seen, but in some regions with resisting skin a verrucose appearance may be presented from cellular infiltration. Minute points or quite extensive areas of gangrene may occur. The bursting of the vesicles and bullæ causes the formation of yellowish or brownish crusts. After the active process in a part has subsided the surface is covered with bran-like scales, large flakes of detached epithelium, and crusts of varied hue. The hair may fall very rapidly, leaving the scalp bare, smooth, and shining.

The temperature-curve follows quite accurately the extension and subsidence of the local process. After the latter has entirely subsided there may remain an elevated temperature, owing to the presence of irritation or actual inflammation of various organs. Cavafy has reported five cases, and I have seen one, of erysipelas of the face without pyrexia.

Not only may the urine contain albumin and an excess of urates, but hyaline and granular tube-casts may also be present. These disappear after the cessation of the disease in the majority of cases. Their presence may be the evidence of the rekindling of a pre-existing disease of the kidneys, in which case they will usually persist or even increase as time passes.

Complications and Sequelæ.—The lung is perhaps the most frequent seat of complication in erysipelas. Pneumonia of the ordinary type is of not

infrequent occurrence, or the specific process may attack the lung-structure. Pleurisy (with or without effusion), empyema, peri- and endo-carditis at times occur. Pleurisy occurred twice in eight cases purposely inoculated by Fehleisen. Previously-existing nephritis is apt to be awakened into activity, and uræmia may be the immediate cause of death. Hæmoglobinuria may be a complication, as in the case reported by Joseph Langer. In facial erysipelas suppurative inflammation of the orbital connective tissue is much to be dreaded, and is frequently fatal from extension to the cerebral meninges through the optic foramen or sphenoidal fissure. Amblyopia or complete amaurosis may result from pressure upon the optic nerve or vessels of the eyeball. Obstinate vomiting is at times a serious complication. Diarrhœa frequently occurs, and the stools may contain blood. After the active signs of disease have disappeared superficial abscesses frequently form.

Erysipelas is, according to Gowers, rarely followed by paralysis. Optic neuritis, optic atrophy, or thrombosis of the retinal vessels may follow compression of the optic nerve and ophthalmic blood-vessels in cases of orbital cellulitis. Amblyopia may be due to retinal hæmorrhages, detachment of the retina, or opacities in the vitreous. In 9209 cases of adventitious deafness analyzed by W. B. Post, erysipelas was the alleged cause in 36.

Diagnosis.—In ordinary cases the diagnosis is readily made. The sudden onset of marked constitutional symptoms coincidently with or rapidly followed by the red, elevated, painful lesion of the skin, the peculiar qualities of the latter, and, in particular, the tendency to spread, sufficiently stamp the disease. When the mucous membranes are first attacked it may be impossible to make a positive diagnosis until the skin becomes affected; but here also the rapid and continuous spread of the disease along the mucous membrane, together with the intense swelling and brilliant redness of the part, should suggest the erysipelatos nature of the inflammation.

Where the poison has entered through the lesions produced by eczema of the hairy scalp, such as is so frequently seen in the neglected children of the poor, the cause of the constitutional symptoms may be only discovered upon the extension of the local process to the forehead, neck, or ears.

From simple erythema the diagnosis is made by the tense swelling, the sharply-defined border, the more marked ambulatory character of the lesion, the fever, and other marked systemic symptoms of erysipelas.

From angio-neurotic œdema this affection differs in all points save the fact of the presence of swelling. From ordinary urticaria it may be distinguished by the rapid appearance and reappearance of "hives," and by the occurrence of the eruption simultaneously in different portions of the body.

The local appearances of acne rosacea sometimes closely resemble those of erysipelas, but the clinical history, the rapidity of extension, and the constitutional symptoms of the latter disease clearly differentiate the two affections.

From malignant œdema the diagnosis must be made by the method of spreading and the local appearances peculiar to the two diseases. Malignant œdema more frequently occurs at points where the skin is particularly thin than does erysipelas.

Prognosis.—In uncomplicated cases the usual result is in complete and rapid cure. In the new-born (that is to say, in those under the age of fifteen days) the disease is practically always fatal, owing in part to the lack of resisting power in those so young, in part to the ease with which extension occurs, and in great part to the liability to the occurrence of phlebitis of the umbilical vein and of peritonitis. In older children complete cure usually results.

Among especially unfavorable occurrences may be mentioned suppuration in the orbital space, gangrene, signs of inflammation of the lung, pericardium, or endocardium. When optic neuritis, optic atrophy, or thrombosis of the retinal arteries occurs, the prognosis as to return of vision is unfavorable. Permanent baldness but seldom results, in spite of the complete alopecia that often is present immediately after the attack.

Treatment.—In this disease the same rules in regard to isolation should be followed as in other contagious diseases, save only in the degree to which it should be practised. Occurring in the medical wards of a hospital, it may not attack other individuals, providing that the beds are in not too close apposition. The contagiousness of erysipelas is not sufficient to warrant the exclusion of cases from medical wards that are properly separated from the surgical and obstetrical departments. It is sufficient that the patient be so placed that he may be surrounded by those having no breach of cutaneous or mucous surfaces. In surgical and obstetrical wards cases of erysipelas should be excluded, and the occurrence of an attack should be the signal for immediate isolation.

No safer means for the prevention of the disease exists than the use of thoroughly antiseptic methods as regards the wards, the operating-room and its appurtenances, the persons of operators and assistants, and the dressings employed. Where attacks recur in an individual any existing lesion that may give entrance to the poison should receive careful and prompt treatment.

In the case of a self-limited disease, and one that rapidly subsides without warning, deductions as to the efficacy of any particular line of treatment must be most carefully drawn. The methods employed in erysipelas are too numerous to be here enumerated; suffice it to mention a few of those that have stood the test of prolonged use by various observers.

A mercurial purgative is advantageous in the early stages and before the institution of any line of treatment. But two drugs deserve mention as having any effect upon the course of the disease—tincture of the chloride of iron and jaborandi. After prolonged trial the first of these seems to have some influence in modifying the severity and shortening the course of the attack. It is best given in large doses, 5 to 15 drops, every three or four hours according to the age of the child. Under its use there is usually found a rapid cessation of extension of the local process and subsidence of the general symptoms. Jaborandi, or its alkaloid pilocarpine, was first recommended by DaCosta, and has had numerous advocates since the announcement of its value in erysipelas. In children, however, it must be given with caution and in doses carefully graduated to the age of the child, the object being to give by hypodermic injection an initial dose of pilocarpine sufficient to produce a pronounced sweat, and thereafter to give every four hours doses of the fluid extract of jaborandi sufficient to maintain a gentle diaphoresis. In adults the method is decidedly beneficial, but in children its use requires caution and careful watching by an intelligent attendant.

The almost purely mechanical rules that govern the extension and limitation of the local process have led to various attempts to substitute artificial boundaries for those of nature. For this end pressure applied in advance of the lesion has been extensively employed by means of tight bandages of elastic material, by the application of strips of adhesive plaster, and by collodion. In many situations no form of pressure is practicable save that by collodion; but the depth to which the constriction by collodion reaches is too slight to offer any obstacle to the spread of the process. Where the other methods are available the application of constricting bandages sufficiently tight to accomplish the object in view is apt to be too painful for their long continuance. As,

however, this does not preclude the employment of other methods of treatment, it should be tried wherever practicable.

Attempts have been made to stay the spread of this specific inflammation by the production of simple inflammatory exudation. For this purpose incisions were made or the solid stick of nitrate of silver was applied to the skin beyond the affected area. Scarification of the healthy skin beyond the edge of the patch has been, and is still, used by some for the same purpose. Hueter first introduced the injection of 2 per cent. carbolic-acid solution under the skin threatened with attack. In some cases it seems to have limited the process, but the method is not always successful. It is, however, rational.

As applications to the diseased area many materials have been recommended, such as flour, lycopodium, or other bland powders, white paint, lead-water and laudanum, cold water, vinegar and water, turpentine, and tar. These are now but seldom used, except white paint and lead-water and laudanum. The exclusion of air of itself seems to relieve much of the discomfort and pain. On this account any emollient application is agreeable. To the fatty base various substances may be added. One of the most agreeable is the hydrochlorate of cocaine in the proportion of 16 grains to the ounce. This usually relieves pain very markedly. Resorcin in the strength of a drachm to the ounce may be used. Koch recommends the application, by means of a bristle-brush, of a mixture of creolin 1 part, iodoform 4 parts, and lanolin 10 parts. Spraying of the affected surface with a solution of corrosive sublimate has been recommended, but greater relief of discomfort, with more likelihood of reaching the deeper parts, can be obtained by the use of constant applications of emollient preparations.

The diet should be nourishing and easily digestible. Milk should constitute the basis during the acute stage of the disease, but eggs, broths, and soft milk foods may be given, except when fever is so great as to interfere with the process of digestion. In all cases occurring among the debilitated, and particularly in very young children, stimulants will be almost invariably required. The amount to be given depends upon the age and condition of the patient.

For extreme elevation of temperature the application of cold externally by means of sponging with cool or cold water, the wet pack, or the cool bath should be employed. Where the hyperpyrexia resists these measures, or where they cannot be properly applied, antipyrine, acetanilid, or, better still, phenacetin, may be cautiously tried. The drugs mentioned should only be employed with extreme care and in minimum effectual doses.

For delirium bromide of potassium or sodium may be given, either by mouth or rectum. Cold applications to the head may be sufficient to moderate the symptom. Opiates are to be used only as a last resource and with great circumspection, not only because of the danger attending their use in childhood, but also because of the liability to insufficiency or actual inflammation of the kidneys in this disease.

Impending suffocation from swelling of the rima glottidis may require tracheotomy. Any purulent collections that may form should be promptly released by the knife.

After the subsidence of the disease tonics with hæmatinics will be required. The alopecia that occurs in some cases usually requires no special treatment, but friction of the scalp and the use of cantharidal preparations will hasten the growth of the hair.

Therapeutic Use.—A few words must be added regarding the use of erysipelas as a therapeutic measure. For many years back there are to be found reports of cases wherein an intercurrent attack of erysipelas was followed by

an amelioration or complete subsidence of the primary affection. The frequency of this phenomenon led to the intentional inoculation of erysipelas for the cure of various affections that were resistant to other measures of treatment, were inaccessible to the surgeon's knife, or whose existence was incompatible with that of erysipelas. Among the affections alleged to have been cured by such an attack of erysipelas or by the intentional inoculation of the streptococcus of Fehleisen may be mentioned various lymphosarcomata, epitheliomata, lupus, and various other chronic superficial ulcerations, keloid, neuralgia, various psychoses, acute polyarthritis, and pulmonary tuberculosis. The antagonism between erysipelas and diphtheria has led to the inoculation of the former upon the latter disease.

While many favorable reports as to the action of erysipelas in the reduction or complete removal of sarcomatous and carcinomatous tumors are to be found, there are others where either no result has been obtained or where recurrence of the growth has taken place, or even death has been brought about by the erysipelalous attack. The cases of neuroses and neuralgia that are found to have been relieved by an attack of the disease can be duplicated by those wherein cure has resulted after many different mental or physical impressions.

In regard to the superficial skin lesions, the favorable action of erysipelas may be explained by the local influence of the inflammation produced as part of the latter. As to the favorable result in a case of pulmonary tuberculosis reported by Chelmonsky, it can only be said that further evidence must be brought forward before any definite curative influence of erysipelas upon this pulmonary lesion can be acknowledged.

Attractive as is the theory of the antagonistic action of the bacterial products in one disease upon its own micro-organisms or upon those of another malady, it seems as yet unjustifiable to purposely add to the existing affection a disease which, while usually ending in recovery, not only may of itself prove fatal, but which is often observed as the final and fatal complication of many long-standing cases of incurable disease.

CHOLERA ASIATICA.

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THIS disease would be most properly designated as *cholera infectiosa epidemica*, for in this term a definite idea of its chief characteristic and of its most marked tendency would be included.

Cholera Asiatica is an exceedingly dangerous specific human disorder, primarily of the digestive tract, occasioned directly by the ingestion, entrance into the small intestine, and exuberant multiplication there of special minute vegetable parasites, the spirilla cholerae Asiaticæ, the so-called "comma bacilli" of Koch. The special poison elaborated by the growth of the parasites in the intestines attacks the epithelial lining of the latter, ultimately reaches the circulation and the nerve-centres, and causes the complex phenomena which characterize the disease.

The intestinal contents, the vomit, and the stools of the attacked contain these specific parasites in enormous numbers, and they are infectious so long as the latter retain their vitality and power of reproduction; so long as their infectious quality persists they are capable, under favorable circumstances, of causing an attack of the same disorder in another exposed, susceptible person, and of giving rise to a local or widespread epidemic of the same disease. For the latter reason does the danger to the public always outweigh in magnitude even that to the individual attacked.

Cholera Asiatica is *endemic* in the lower two-thirds of the presidency of Bengal, roughly corresponding to the delta of the Ganges and the Brahmapootra; it becomes *epidemic* in other parts of Hindostan and of the world only periodically, after more or less irregular intervals of entire absence. During the intervals of epidemics, except as scattered cases shortly preceding or following such visitations, and as an essential part of the latter, it does not exist outside the endemic area: it has no more affiliation with or relation to our somewhat common so-called summer cholera—otherwise termed *cholera nostras*, *cholera morbus*—than it has with some acute attacks due to arsenical poisoning, to ptomaine-poisoning from ingestion of decomposed food, or to acute pernicious malaria, or to still other very different disorders, all of which, nevertheless, not infrequently present very similar symptoms and terminations.

Etiology.—Although abounding filth of the surroundings—that is, of the district or the locality, of the domicile, of the home-life, and of personal habits—favors infection and the subsequent development of an individual attack, and the initiation, continuance, and spread of an epidemic of cholera Asiatica, neither a personal seizure nor an epidemic outside that endemic area which is the natural home of this disease can occur (not even when the person or population wallow in every sort of reeking abomination), unless the special infection be first introduced. In other words, no amount of filth is capable of producing a *spontaneous generation* of the specific infection which is the active

cause of this disease; nor, without the activity of this specific cause, is any other agency or influence capable of producing the disease.

The active specific cause of cholera Asiatica is the presence and multiplication in the intestinal canal of the subject of numbers of very minute vegetable parasites, certain well-defined species of bacteria known as the spirillum cholerae Asiaticae discovered by Koch in 1883, and because of their usual resemblance under the microscope to the written *comma*, and of the name of its discoverer, commonly called the "comma bacilli of Koch."

The term "bacillus" as applied to this vegetable micro-organism is, however, a misnomer, for the species is now regarded by nearly all competent authorities as a member of the group of spirilla. As commonly encountered in the intestinal contents or vomit of a victim of the disease, and in artificial culture media when growth is recent and rapid, if a fresh preparation be placed under a microscope of very high power and excellent definition, this micro-organism is usually so actively mobile as to defy distinct vision. If the fresh preparation has been made from a recent pure culture, and there be plenty of fluid under the thin cover-glass, the movements of the comma bacilli remind one of the rapid, darting, zig-zag movements of the individuals of a swarm of small flies, and of the impossibility of distinct vision of any one of the swarm. If, however, a smear-preparation from such a culture be made, and after drying and flaming in the usual manner, this be properly stained, mounted, and examined, it will be seen that each form is more or less curved—a few almost imperceptibly so; a few others nearly as much as a semi-circle; the greater number having a curvature representing an eighth or a quarter of a circle. The length may vary from one-seventh to one-fourth the average diameter of the red blood-corpuscle of man, the width being about a fourth its length. Examined critically it can often be seen that, instead of forming a segment of a circular ring, the individual form is in reality a portion of a spiral. The ends are blunt but rounded, sometimes slightly tapering, then presenting an outline similar to the fennel-seed. When proper methods of staining are used each end of the "comma bacillus" is found to be furnished with one or more flagella, which act as motive organs. Cultivated in bouillon by the hanging-drop method, besides the above-described forms there are usually seen a variable number of more or less long and complete spirilla. Old cultures in bouillon, in gelatin, in agar, and in other media nearly always contain the comma and spiral forms, and intermingled with these are frequently other shapes, which many authorities regard as involution forms. Chief among the latter are spherules of a diameter from that of a cross-section of the comma to that of a red blood-corpuscle of man, and even greater. It is pretty certain that neither the comma nor the spirillum forms contain spores; vacuoles have been mistaken for them. In the vomit and intestinal contents of the attacked the comma forms are always present for a number of days, and short and incomplete spirils may sometimes be demonstrated in smear-preparations.

The comma bacillus of Koch multiplies commonly by two modes, each of which, however, constitutes essentially a process of fission: *a*, the comma doubles its length, and then divides into two; *b*, before dividing the comma continues its elongation into a longer or shorter spiril filament, which ultimately becomes segmented in order that finally the segments may separate to form new and separate commas. Of these two processes of multiplication, the former is by far the more rapid. Elongation and division of the one comma into two have been actually observed under the microscope to take place in twenty minutes. With such a rate of multiplication demonstrated, one can easily form some adequate conception of the otherwise inconceivable rapidity of

PLATE VIII.

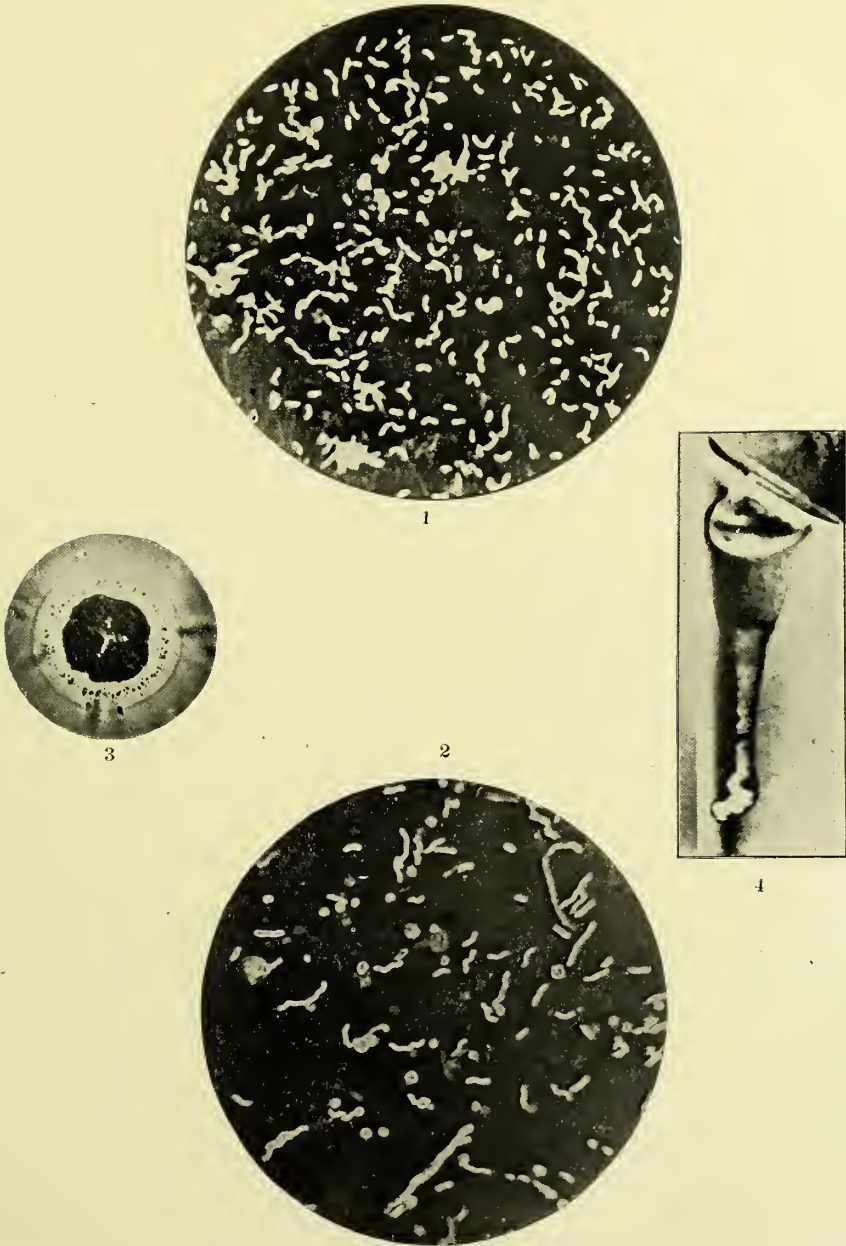


FIG. 1. Photo-micrograph : Smear preparation from pure culture of comma bacillus of Koch. $\times 1200$.

FIG. 2. Photo-micrograph : Smear preparation from (old) pure culture in gelatin of comma bacillus of Koch, showing oögonia of Ferrán or involution forms of other authors. $\times 1200$.

FIG. 3. Photo-micrograph : Gelatin-plate colony of comma bacillus of Koch. $\times 50$.

FIG. 4. Photograph : Gelatin tube-culture of comma bacillus of Koch, 72 hours old, surface inclined. Natural size.

propagation and enormous power of dissemination in river-water of the specific infectious principle of Asiatic cholera contained in the discharges from the bowels of a few cases, numerous examples of which the history of this disease affords; one of the most striking being the most recent—namely, that of the river Elbe in 1892. Of other possible modes of multiplication, only two may be merely mentioned here: that by intervénion of so-called arthrospores of Hüppe, who claims that these reproductive bodies approach the tenacity of life and the power of resistance of genuine spores; and that of so-called “ögonia” of Ferrán—both modes being a form of multiplication by budding.

The multiplication of the comma bacillus of Koch in artificial culture media has been found to vary greatly under different constitution of media and varying conditions of temperature, etc. During the development and continued growth of these organisms in artificial culture media, chemical combinations are split up and various new chemical products formed, as the necessary accompaniment of the nutrition, life, or death of the microbes; and these resultant new chemical products vary in quantity or composition, or both, with the varied chemical and physical complexion of the culture media, the external conditions of temperature, moisture, free oxygen, light, etc. Thus it seems to be now pretty clearly established that in artificial culture, among many other characteristics, the cholera microbe will not develop at a temperature below $57\frac{1}{2}^{\circ}$ F. or above $107\frac{3}{8}^{\circ}$ F.; that freezing, unless it be prolonged, does not kill this microbe, but places it in a state of hibernation, as it were, ready to resume again all its vital and pathogenic functions with the return of sufficient heat; then, on the contrary, when a temperature of $107\frac{3}{8}^{\circ}$ F. is exceeded the vital functions of the microbe are more and more inhibited permanently, if the temperature be continued, until a point is reached, at about 140° F., where the life of the microbe is destroyed absolutely in a very few minutes; that multiplication is more rapid in fluid media of suitable constitution; that the culture fluid, as a rule, possesses more virulence when the inoculated microbes are very recently obtained from an active case of cholera than when a long time has elapsed; that the presence of peptone in the culture medium seems to materially increase the development of the virulent power of the microbe, especially when free oxygen and light are excluded; that there is scarcely any fluid or solid moist nutrient material of animal or vegetable composition, of a neutral or slightly alkaline reaction and not containing a substance possessing antiseptic properties, upon or in which it will not grow; and there are at the same time many fruits and vegetables upon the pulp or surface of which the microbes of cholera will not only live for hours and days, but will multiply there even when the object gives a slightly acid reaction. This microbe will live and multiply enormously for a time in pure water, in foul water, even in sewerage, and in sea-water; it will live for a considerable time and multiply enormously in milk, whether fresh or previously sterilized; it is capable of living and multiplying for a time in various common beverages and on various common articles of food. It will retain its vitality, sometimes multiply exuberantly, on various textile fabrics of vegetable or animal nature for days, and in some cases weeks and even months, if they be not thoroughly desiccated or exposed to the sun's rays, and contain no antiseptic substance; if such fabrics be kept decidedly damp or wet, the germ is capable of enormous multiplication, and of retaining its infectious and reproductive power to a virulent degree for indefinite periods, lasting for weeks or months, provided the sunlight does not fall upon it. If, however, these fabrics are thoroughly dry before the microbe is placed upon them, and remain or quickly become thoroughly dry afterward, it soon dies—more quickly still if exposed to the sunshine or bright reflected light. Whilst the propagative power of the

cholera microbe outside the human body, under favorable circumstances, is so enormous as to be almost incredible, fortunately for man it is, of all the dangerous pathogenic microbes known, the most susceptible to restraining or destructive influences. Whilst it is too often true that an individual, a community, a city, a whole nation, or even a continent, presenting favorable conditions for the free propagation of the infection, oftentimes suffers consequences which in their swiftness, gravity, and manifold relations may be appalling, yet there is no infectious epidemic disease which can so certainly and so easily be warded off or arrested as can Asiatic cholera.

Mention has already been made of the ingestion, entrance into the small intestine, and exuberant multiplication there of the "comma bacillus of Koch" as necessary conditions precedent to an attack of Asiatic cholera. Even with these it is probable that there must be one more condition before a serious attack follows—namely, susceptibility to the disease on the part of the individual. Since desiccation is one of the sure and rapid means of killing the microbe of cholera, and since the comma bacillus does not exist in the lungs or intestinal organs, in the blood, lymph, or muscular tissue, or in the nervous system of a person suffering an attack of cholera, it is obvious, *a priori*, that the active infection of this disease is neither inhaled nor does it enter through the cutaneous surfaces. But in this matter we are not obliged to rely upon inductive reasoning, for there is not a single example known of either mode of infection in the clinical history of cholera or in laboratory experience with this disease. The cholera microbe must be *swallowed* and pass from the stomach into the small intestine alive and endowed with vigorous powers of propagation and pathogenesis, before cholera can be naturally produced in man.

There are various means and modes by which the infection of cholera may be introduced into the œsophagus of man. It may be conveyed by various fluids imbibed, such as water, milk, beer, weak tea, etc.; by various articles of food, such as raw vegetables, bread, butter, fruits, meats, etc.; by contact of the mouth with hands in some way soiled through careless handling of objects contaminated with numbers of the microbe, such as the clothing worn by the sick, the bed-linen used by them, the vessels containing the vomit or stools, etc.; by water used for lavatory purposes or the washing of dishes or other food-receptacles; by water used for washing the mouth and teeth, etc. The corollary of all this is that Asiatic cholera is not acquired by inhalation or mere contact with persons suffering from the disease, or with things contaminated with the infectious principle. Moreover, there seems to be a natural insusceptibility on the part of many to an attack of cholera, although they be undoubtedly exposed to the infection. Numerous examples of this personal immunity are furnished by every great epidemic, especially when the outbreak has been caused by contamination of the common supply of drinking-water. Furthermore, there is incontrovertible evidence to prove that there is an acquired immunity of variable duration following a natural attack of Asiatic cholera, whether the latter have been grave or mild. Indeed, it is pretty certain that a natural attack so light as to have escaped recognition is capable of producing such an immunity. That an immunity can be acquired artificially by means of inoculations of various kinds and in various ways now seems to be an established fact. I need only mention in this connection the pioneer work of the Spanish physician, Dr. J. Ferrán in 1884 and 1885, and after him the investigations of Petri, Brieger, Wasserman, and Kitasato, Klemperer, Klebs, and Hafkine, which with those of others constitute a body of experimental data so convincing as to leave but little, if indeed any, room for reasonable doubt. Whether or not an attack of cholera follow introduction of the

special *contagium vivum* into the stomach of man may depend upon one or more of several conditions. The acid gastric juice of the stomach is, when present in sufficient quantity relative to the number of cholera microbes, capable of quickly killing them. Hence at times when the stomach is properly functioning and the number of the cholera bacilli swallowed is not excessive, there is far less probability of these microbes passing the pylorus alive and still retaining their vigorous pathogenic powers than when either there is little or no acid in the stomach or but little relative to an excessive number of comma bacilli introduced. Then, again, the factor of personal susceptibility—or, if we prefer its complement, we may say the factor of personal immunity—may intervene (after the cholera microbes have passed into the small intestine alive, virulently pathogenic and in sufficient numbers, with certain limitations), either to render an attack of cholera more certain of development and more violent, or to prevent it entirely, or to render it milder, respectively, as the case may be. Thus there is strong reason to believe that in Asiatic cholera as in other infectious diseases, whether the degree of susceptibility or the degree of immunity of any person be great or little, the dosage of the infectious material is a matter of importance for the generation or the violence of an attack. Any degree of immunity can be overwhelmed by an excessive dose, and any degree of susceptibility can be rendered insufficient by too small a dose. These considerations explain why it is that of so many exposed to the infection of cholera only a comparative few suffer an attack which is recognized as such. They also explain why a few foolhardy persons, whose skepticism seems to be greater than their power of discrimination, have ostentatiously swallowed voluntarily, in former times, some of the intestinal discharges of cholera victims, and in later times, some quantities of pure culture of the cholera microbe, and have lived to preach their false doctrine.

When a sufficient number of vigorous pathogenic cholera microbes is introduced into the stomach and passes with vital properties unimpaired into the small intestine of a susceptible person, an attack of infectious cholera may be developed. In such a case the cholera microbes multiply enormously, and often with great rapidity, in the small intestine. With their growth there, under favorable conditions not yet well determined, a virulent specific chemical poison is generated. Whether this poison be essentially a *ptomaine* analogous to the highly-poisonous vegetable alkaloids, as some contend, or a species of virulent *albumose*, as others maintain, or a special pathogenic *enzyme*, as a few affirm, or possess other characteristics, or be a combination of two or more of these, it would be unprofitable to discuss in this place. Whatever the nature of this specific chemical poison may be, it is pretty certain that when generated in sufficient quantity it attacks primarily the epithelium of the mucous membrane of the small intestine, exciting in it the phenomena of irritation and degeneration in varying degrees—according to the concentration of the poison and the susceptibility of the person—from initial cloudy swelling all the way to complete fatty degeneration and desquamation. The irritant poison penetrates beyond the epithelium and excites in a susceptible person a round-celled infiltration of the connective tissue underlying the epithelium; it may even exert its irritant powers upon the submucous layer of connective tissue, and sometimes its influence may even extend outward into the muscular and subserous coats of the intestine calling forth in them varying inflammatory phenomena. Klebs pointed out that autopsies of rapid cases of cholera showed invariably the inner surface of the small intestine to be covered with a very tenacious coating of mucus, and the experience of most observers confirms him. Another characteristic is that the serous mem-

brane of the small intestine is likewise the seat almost always of a viscid covering, consisting mainly of degenerated and proliferated endothelium. The inflammatory action in the mucous and submucous coats of the small intestine may become so intense as to result in more or less extensive necrosis. Very generally the mucous membrane is hyperæmic. This hyperæmia may be very diffuse or it may be limited to larger or smaller areas. It is usually most marked in the region of the ileo-cæcal valve and around the Peyer's glands. The Peyer's glands and the solitary follicles are usually infiltrated and prominent, and this is so common that some French authors have regarded cholera as a specific psorenteritis. The infiltration of these glands may in some instances be so intense as to end in necrosis and ulceration. Notwithstanding the fact that the chemical poison of cholera attacks locally, first, the intestinal epithelium, and then the subjacent layers of connective tissue, sometimes even to the point of denudation and limited destruction of the latter, the cholera microbe itself never penetrates the coats of the intestine except when they are denuded, and then does not pass beyond the most superficial portion of the exposed connective tissue: it never enters the lacteals or reaches the general circulation. The chemical poison, however, which is produced in the intestinal canal by the growth of the cholera microbes therein, does not limit its action to a local attack upon the intestinal epithelium or upon the subjacent tissues; but it is taken up by the intestinal absorbents or the capillaries of the villi, and enters the general circulation of the blood to be distributed to every organ and tissue in the body, to develop in the susceptible its secondary or constitutional action. It may be said, therefore, that cholera infectiosa epidemica is essentially a specific systemic intoxication. It may not always happen that the whole or the greater portion of the specific poison which produces an attack of Asiatic cholera has been generated within the intestinal canal of the victim; there is strong reason for the belief that exceptionally, at least, the offending material ingested already contains, before swallowing, a sufficient quantity of the specific chemical poison of cholera to produce an attack of the disease. It is probable that at least some of those attacks with a violent onset in a very few hours after exposure to the infection have resulted in such a manner, especially if the autopsy show, as it sometimes does, very little alteration of the intestinal mucous membrane. I can conceive, for example, how milk diluted with water contaminated with cholera dejecta, and then allowed to stand for several hours in a warm place, can act as a quick and fatal poison when swallowed in large quantities. In such a case it would matter not if the bacteria were killed in the stomach by the action of the gastric juice; the preformed chemical poison of cholera when absorbed from the intestine and circulated in the blood might, if in sufficient quantity, still be capable of causing a violent, and even a mortal, attack of cholera. The stools from such a victim of the cholera poison might still contain some quantity of that poison, but could not, in the absence from them of the living pathogenic comma bacillus of Koch, be infectious. In other words, from such a victim a new case of cholera could not arise, much less an epidemic. Furthermore, although the symptoms, course, termination, and post-mortem appearances observed in such a case would naturally be those characteristic of cholera, yet a culture test of the stools would necessarily be negative in result, and therefore misleading as to the origin of the attack, if not, indeed, of its nature. *A priori*, it is just among young children, who consume habitually large quantities of milk, that we should look for the largest proportion of such toxic non-contagious attacks of cholera.

Symptoms.—For convenience of description in part, and in part also

because the common course of the attack furnishes the basis of the division, clinical writers have been in the habit of discussing the symptoms of Asiatic cholera under four periods: *a*, the prodromal period; *b*, that of serous evacuation; *c*, that of algidity or collapse; *d*, that of reaction.

a. The prodromal period, or period of incubation, varies in duration from a few hours to perhaps five days. Probably its average length may be most accurately reckoned at forty-eight hours. It is the time which elapses between the ingestion of the infectious material and onset of pronounced symptoms. During the early part of the period, sometimes during the whole of it, the subject is apparently in his accustomed health, whilst in the latter part of it, and occasionally throughout its entire length, and increasing in severity toward its transition into the next period, there may be a general feeling of distress in the abdomen, or even a tendency to nausea, with or without tenderness, restlessness, rumbling, and increased peristaltic movement of the intestines sometimes visible or palpable through the abdominal walls; laxness of the bowels or decided diarrhoea, with colored semifluid, feculent, or decidedly fluid, usually painless, sometimes copious, evacuations. All of these symptoms may be present, or only one of them, or they all may be absent. There is nothing at all distinctive in their character which is in any way suggestive of their special nature. They excite suspicion only when it is known or suspected that the person may have been exposed to the infection of cholera, or when the disease is present in the locality. There is no indication of systemic intoxication during this period. The cholera microbe has merely reached the small intestine, and is more or less quietly gathering its forces for the active attack. It is engaged in multiplying itself and in generating its specific poison. The assault on the epithelial lining of the small intestine may have actually begun, and some breaches in its integrity have been accomplished; sufficient of the chemical poison may have been generated for the production of some hyperæmia of the mucous membrane, or even for the excitement of some infiltration of the subepithelial connective tissue; but there has been as yet no systemic absorption of the specific chemical poison; the action of the special poison is still local, although there may be experienced a degree of prostration out of all proportion to the diarrhoea present.

b. The period of serous evacuations may be regarded as that of systemic intoxication, and its duration may last from a few hours to a day or two. The prodromal diarrhoea, if it have existed, now usually assumes more gravity. The discharges become more frequent, copious, and fluid. Often, but not always, every trace of color disappears from the stools. The latter now frequently present the well-known rice-water aspect: they are thin, very watery, and hold in suspension more or less minute whitish flakes or shreds in great numbers; they look like a watery gruel, in fact closely resemble the aspect of barley-water or macaroni-water. They may sometimes still be slightly colored, and they are not infrequently frothy or somewhat bloody. In fact, there is many a case of cholera Asiatica where the stools are bilious or lack entirely the familiar rice-water appearance. Often the desire to evacuate the bowels is sudden and absolutely uncontrollable, and the contents of the lower colon and rectum are sometimes expelled with great force without pain and in enormous quantity, saturating the bed and covering, or deluging the clothing if the patient be still up and moving around. Nausea and vomiting are now usual accompaniments. At first the vomit may be bilious; later it assumes the rice-water or gruel aspect. The amount of fluid discharged from the anus and mouth is often excessive. Prostration quickly becomes extreme, and thirst intense. The cry for water is constant, yet it is rejected by the

stomach almost immediately after it is swallowed. The enormous exudation of fluid into the intestinal canal reduces correspondingly the volume of the lymph in the tissues and organs, and of the blood in the circulatory system. The tissues become abnormally dry and shrunken, and the blood markedly thickened. The number of the corpuscles of the blood is relatively much increased per cubic centimetre; it is sometimes nearly doubled. The heart has not of itself the power to propel this thickened fluid with sufficient vigor to prevent venous stagnation. At first the pulse is very frequent for a time; indeed, palpitation may add to the general distress and anxiety of the patient; besides being accelerated, the pulse is usually at the same time small, feeble, and soft. Later the heart's action becomes more and more enfeebled, until the pulse is nearly or quite lost at the wrist, whilst the apex-beat may also nearly or quite disappear, and the heart-sounds themselves decidedly change their character—the systolic sound being greatly weakened, or even replaced by a faint blowing murmur, and the second sound lost entirely. The loss of fluid is shown in the deeply sunken orbits, glazed corneæ, the pinched expression of the face, the wrinkled condition of the palmar surface of the hands and feet—the washer-woman's hands—and the general emaciation, which often becomes extremely marked. The impeded circulation of the blood is evidenced by the more or less lividity, which is most marked around the eyes, the ears, the lips, and the ends of the fingers. The surface temperature sensibly falls below the normal, sometimes markedly; on the contrary, the rectal temperature is usually considerably above the normal. The temperature under the tongue is commonly subnormal, and the tongue itself often feels cold to the touch. Whilst the cutaneous surface is objectively cold, the patient himself will frequently complain of intense internal heat. The voice becomes weak, hollow, and husky. The intellect may be clear or clouded. Sometimes there is great restlessness and jactitation; at other times there may be entire calm and hebetude approaching to stupor. Oftentimes cramps in the extremities and trunk may be absent or mild and fleeting, or they may be so violent as to cause agonizing pain to the patient. In the early part of this period there is marked diminution of urine associated with albuminuria, and frequently, granular tube-casts. Very soon, however, secretion of urine is completely suppressed. While the blood is robbed of chloride of sodium and serum by the exudation into the intestinal canal, it is overladen with urea, which the kidneys fail to remove, and there is proportionately more of its salts in the central nervous system than anywhere else in the body.

We have said that this period should be regarded as that of systemic intoxication. The specific chemical poison elaborated in the small intestine during the enormous multiplication of the comma bacillus of Koch, has at length been taken up by the intestinal absorbents or has entered the network of intestinal capillaries, and has reached the general circulation of the blood. From this moment the scope of its action is no longer localized in the small intestine, but is now extended throughout the whole system. The presence of this specific poison in the blood of the susceptible, works changes in the complexion of this vital fluid, some of which are readily visible. We have already spoken of the relative increase of the corpuscular elements due to loss of fluid. There is, however, a material change in the red corpuscles, probably due to the effect of the special chemical poison: many of the red corpuscles are much paler than normal, and also much smaller; some have been broken up into very small particles, which by reason of their form and frequent arrangement in pairs and chaplets have been mistaken for micrococci. The specific gravity of the blood is much increased; there is little or

no tendency of the red corpuscles to adhere together, and there is little tendency to the formation of large clots when allowed to stand; if there be any separation of serum, it is very slight. The blood when drawn from the veins is very dark, almost black in color and tarry in consistence.

This abnormality of the blood does not, of course, reach its height at once with the commencement of this stage, but progresses with the continuance and severity of the exudation of the fluids into the intestinal canal during this period. The blood becomes so thick and the heart's action so weak that the flow in the veins becomes exceedingly slow or seems to be arrested entirely toward the end; it sometimes will not flow from an incision. The left side of the heart may contain but little blood, and the large arteries, which are often spasmodically contracted, are nearly empty. The right side of the heart, on the contrary, is full oftentimes to over-distention. The lungs are usually found, post-mortem, to be quite pale, bloodless, and retracted well against the spinal column. In the mesenteries the arteries are much contracted, while the veins are greatly dilated, and there is usually also capillary engorgement. In fact, this condition of strong contraction and emptiness of the calibre of arteries, wide dilatation and fulness of the veins and capillaries, is observable nearly everywhere. There are often also small ecchymoses, and sometimes rather extensive extravasations, particularly at the mucous surfaces. Edemas, however, are not to be met with; notwithstanding the numerous stagnations of the blood-current in veins and capillaries, the flow of fluids of the blood into the intestinal canal is so great, and the consistency of the blood has become so thick, that everywhere else than at the mucous surface of the intestines the tendency to fluid exudation has been completely arrested. The ecchymoses above mentioned are more abundantly scattered over the mucous and serous surfaces than elsewhere, although they may exist even in the muscular tissue.

The toxic influence of the specific chemical poison in the blood is probably most marked upon the central nervous system (including the sympathetic ganglionic system), and upon the liver and kidneys, especially the latter. The mechanical results of loss of such an enormous quantity of body fluid may in some part account for the seriousness and severity of the symptoms of this and the following period; but doubtless the action of the chemical poison in the blood upon the nervous system, the liver, and the kidneys is even superior. The first onslaught of the poison upon any important internal organ after reaching the blood naturally falls upon the liver. This organ is generally smaller than normal, flaccid, and anæmic, and contains less glycogen than normal. The outlines of the lobules are more or less indistinct; the interlobular network of blood-vessels may or may not be dilated and filled with blood; the radiating cellular trabeculæ of many lobules are decidedly narrowed, while the intertrabecular blood-capillaries of some portions of acini are dilated and filled with blood-corpuscles. The hepatic cells of many acini are granular and difficult to stain. Some investigators contend that there is actually some atrophy of the liver. The gall-bladder, the cystic and common ducts are distended with a thin brownish or greenish fluid, whilst the interlobular biliary network is not appreciably altered. Whilst the biliary ducts and gall-bladder are full, the intestinal end of the ductus communis choledochus is usually practically impermeable, and the intestines rarely contain any bile. The spleen is contracted and often flabby. Next to the intestinal lesions in cholera the kidneys show the greatest pathological changes. The effect of the cholera poison in the blood falls heavily upon these emunctories. Granular degeneration of the secretory tubules of the cortex soon becomes marked, but is irregularly distributed at first. After this pathological process has continued for some time,

fatty degeneration of the tubular epithelium becomes general and intense, and associated sometimes with parenchymatous inflammation. The suppression of urine is therefore not alone due to the mechanical effects of thickening of the blood.

c. The period of algidity or collapse may follow after a few hours of continuance of the period of serous evacuations, and may last for some or many hours until death or reaction ensues. In this desperate condition prostration is extreme; the voice is gone; respiration is very feeble, shallow, and fitful; the pulse has vanished and the heart almost ceases to beat; so also the nausea, vomiting, and cramps, the frequent enormous forcible evacuations of the bowels, whilst, instead of the latter, the contents of the intestines dribble away from the anus, whose sphincter is inactive. Profound stupor or coma is the rule. The general lividity is intense; the coldness of the skin is like that of marble. The vital forces are nearly overwhelmed by the great losses of fluid sustained, by the effete substances which are accumulated, and by the special cholera poison. During this period the vital spark flickers very faintly; life hangs trembling in the balance. The pathological conditions are essentially those of the previous period, intensified.

d. The period of reaction may be short or prolonged, and directly follow either of the three preceding. It may last from three or four days to as many weeks. When it follows immediately upon the prodromal period, convalescence is usually rapid and short, and the wonted health is soon perfectly re-established. In such a case there is, after all is over, of course, great doubt that the attack was choleraic at all. The finding of the comma bacilli of Koch in the stools is the only certain criterion of what its true nature has been. When the period of reaction immediately follows the period of serous evacuations, it is usually the more definite the more serious the symptoms and pathological lesions during the latter period have been. If there have been great alterations of the mucous membrane of the intestines, profound general intoxication, with great destruction of the red elements of the blood and marked degenerations in the liver and kidneys, we may expect to witness a more or less prolonged, complex, and dangerous period of reaction. In fact, as a rule, more patients die during than before reaction, when the latter follows immediately the period of serous evacuations. The gravity of the symptoms and general condition of the patient may slowly ameliorate or quickly improve, or one set of alarming symptoms may simply be substituted by another set, which, although not so frightful to the laity, will be regarded by the experienced physician as only a prolongation of the critical struggle between the very evenly balanced forces of life and of death. The evacuations from the stomach and bowels decidedly lessen in frequency and copiousness; the stools lose their barley-water aspect; the bile reappears in them, and they assume gradually the common characteristics of an ordinary diarrhoea, sometimes stained with blood; or if the local destructive effects of the cholera poison have been drastic, there may be grafted upon the diarrhoea a more or less pronounced dysenteric condition with bloody stools and tenesmus. The characteristic aromatic sperm-like odor of the rice-water stools may now change to the foul, stinking odor of decomposition, and the flatulence which was absent during the preceding period may become annoying. The voice becomes stronger, respiration more steady and fuller. The heart gradually regains its lost powers; the pulse begins again to be felt at the wrist; the surface temperature again goes toward the normal and quickly passes above it; the shrunken countenance begins to discard the Hippocratic expression, the sunken orbits to fill up and the glazed eyes to brighten; prostration becomes less marked,

thirst less intense; the secretion of urine is slowly re-established, at first containing much albumin, granular casts, and large quantities of urea; appetite and digestion are slowly recovered as a rule. In fortunate cases the restoration to health and to the proper exercise of all the bodily functions may be rapid and complete. But in other cases anæmia, due to the great injury to the elements of the blood, may be protracted; or the functions of the much-damaged kidneys may be slow of re-establishment; or the destruction of intestinal epithelium may leave denuded patches in the subepithelial layers of connective tissue, and thus occasion prolonged irritation and even serious derangement of the processes of digestion, and at the same time furnish numerous points of entrance for various septic micro-organisms. In truth, a secondary septic fever, as the result of systemic invasion in this manner, is not at all uncommon in this period: it is vulgarly called the typhoid stage of cholera.

When the patient passes through the period of serous evacuations and that of algidity or collapse, the period of reaction usually differs only in degree from the condition above described. It can be now readily understood why almost as many victims succumb during the period of reaction as during the periods of specific action of the cholera poison. Even after convalescence has been established impaired health may persist for a long time, evinced by chronic anæmia, stubborn disorders of the digestive apparatus, and easily disturbed bowels. Before convalescence is fully confirmed, and even for some time afterward, imprudences of diet sometimes precipitate a dangerous relapse.

Special Phases of Cholera.—In a virulent epidemic of cholera the cases of very sudden and violent attacks, which do not seem to have been preceded either by a prodromal period or the one described in section *b*, are sometimes numerous, and they are most frequently encountered near the commencement of the outbreak. These attacks have been variously named foudroyant, toxic, asphyxic. In description of these foudroyant attacks we cannot do better than quote the recent language of Dr. N. J. Simpson, the health officer of Calcutta: "On these occasions the suddenness of the attack, the number affected, and the virulence of the disease would incline one to think that the specific organisms had already elaborated outside the human body a strong poison which acted on the victim almost immediately after being swallowed. Under the most favorable conditions for the elaboration of such a poison there will not, as far as can be ascertained, be the usual twelve to forty-eight hours' period of incubation; on the contrary, patients will be brought into hospital in a dying state, though taken ill only a short time previously; some will die before reaching the hospital; and the ratio of mortality is likely to be 75 to 85 per cent. The description given by Dr. Jamieson in 1817 seemed to me until some time ago somewhat exaggerated, when the cases seen during an outbreak at a large pilgrimage convinced me of the correctness of Jamieson's accounts as applied to exceptional outbreaks. He says: 'Sometimes there was no vomiting, sometimes no purging, sometimes no spasm throughout, sometimes all these symptoms were simultaneous, and the vomiting and purging took place together, as if caused by sudden contraction of the alimentary canal in its whole extent. In some rare cases the virulence of the disease was so powerful as to prove immediately destructive to life, as if the circulation were at once arrested and the vital powers wholly overwhelmed. In these cases the patient fell down as if struck by lightning, and instantly expired. Others, again, sank after making one or two feeble efforts to vomit and drawing a long and anxious inspiration; some recovered from the insensibility produced from the first shock, and afterward went through the regular course of

the disease.' In these and similar cases a virulent poison is the best explanation of the symptoms and apparent absence of the period of incubation, and of the destructive nature of the disease." Another phase of cholera still more rarely met with is what has been termed *cholera sicca*. In this there is no vomiting, no purging, but the other symptoms may be little different from those already described. The autopsy shows, however, that there has nevertheless been great exudation of fluid into the intestinal canal, for the latter is greatly distended with it from end to end.

Special Complications of Cholera.—I have already spoken of frequent occurrences of ecchymoses, especially on the mucous and serous surfaces. Cutaneous petechiæ and eruptions are not uncommon in the period of reaction; they appear less frequently during that of serous evacuations or algidity. These eruptions, more often observed on the face, neck, and forearms than elsewhere, are usually more or less punctate, the puncta being slightly elevated and having a tendency at times to aggregate into irregular groups. These spots vary somewhat in color, but most frequently the points are dark or black. In some rare cases the vitality of the skin seems to be in a degree impaired, as indicated by a disposition to ulcerate upon small provocation; for example, bed-sores may sometimes develop early and become an exceedingly troublesome complication. The cause of these eruptions is unknown, but if we were to express a mere conjecture, it would be that they may be due to innumerable minute thrombi and emboli—small clots which have formed during stasis of the blood.

Diagnosis.—The differential diagnosis of Asiatic cholera by means of its symptoms alone is, during the absence of an epidemic of the disease, one of the most difficult feats the clinician is ever called upon to perform. Indeed, it is held by some of the most skilful and renowned clinical diagnosticians in the world to be an utter impossibility to make a certain diagnosis; and it is, and always has been, the common experience of the whole world that the saddest, and for the public health the most deplorable, mistakes are very often made even by the most experienced. And yet there is no single one of the whole category of diseases with respect to which a mistake in diagnosis of a first case may, and sometimes does, entail such an endless series of incalculable public calamities. There is not one of the symptoms, and of the groups of symptoms, met with in some period of an attack of Asiatic cholera, which does not perfectly resemble those of some disease which is more or less common. Among these commoner affections for which Asiatic cholera may be mistaken clinically are cholera morbus, arsenical poisoning, pernicious intermittent fever, and poisoning from consumption of various articles of food in special states of decomposition or fermentation.

Of course during the prevalence of an epidemic in a locality, the physician of that place will wisely regard and treat every case presenting the symptoms common in Asiatic cholera as an undoubted case, and will not hesitate to handle it as such; for the community will unquestionably uphold him. It is, however, just when the physician is most uncertain—namely, in dealing with those doubtful cases which precede and follow the epidemic—that the real interests of the community and of the general public demand the greatest certainty of diagnosis; but then, as a rule, the people are unwilling to submit to restraints. Fortunately, through the discovery of Koch in 1883 and 1884, we now possess the means of making an absolutely certain differential diagnosis of cholera infectiosa epidemica, and without reliance upon clinical symptoms, which may be misleading, or upon trustworthy knowledge of the previous history or relations of the patient, which may be difficult or impossible to obtain. The

presence or absence in the stools of the suspect of the comma bacillus of Koch promptly and definitely settles the matter. This can be determined within forty-eight hours by resort to the microscopic and biological tests. These tests, however, should never be relied upon when made by a tyro. They are too difficult of application to be trusted to the inexperienced. To describe here the methods of procedure would therefore be useless, for the experienced bacteriologist does not need such instruction, whilst the unskilled would need much more to be rendered capable. During times of great danger of the introduction of Asiatic cholera into a locality all cases presenting the symptoms of cholera should be handled as suspicious until a differential diagnosis by means of the microscopic and biological tests be made by a thoroughly competent and experienced bacteriologist.

Prognosis.—The outcome of an attack of cholera depends very much upon what period of the seizure medical advice is had, very much upon the slowness or rapidity with which grave symptoms appear and persist, very much sometimes upon the period of the epidemic at which the attack happens, and very much upon the constancy of intelligent care in handling the case from first to last. Wise and prompt treatment of the first stage usually aborts the attack almost in the beginning, and is followed by scarcely any mortality. In the vast majority of such cases the attack never gets beyond the stage of premonitory diarrhœa, and convalescence is usually rapid and complete. The prognosis of a seizure which has passed into the second period, or that of pronounced serous diarrhœa, is grave; the mortality varies greatly, from 25 to 60 per cent. of attacks, by reason of the varying susceptibility of patients, varying doses of the specific poison, varying promptness, persistency, and wisdom of treatment. The prognosis of an attack of Asiatic cholera in the period of algidity or collapse is truly desperate, and the mortality has usually been frightful, not infrequently having reached 80, 90, and sometimes 100 per cent. The prognosis of an attack which has reached the period of reaction varies greatly according to the damage which may have been done the intestinal lining, the secretory elements of the kidneys, the glandular elements of the liver, and the elements of the blood, and in proportion to the accumulations of effete material and of specific poison in the blood and tissues. It is sufficiently serious to require careful nursing and wise medical direction; where septic poisoning has been engrafted upon the cholera attack, it is often grave. Speaking generally, the mortality of epidemics of Asiatic cholera is usually greatest in the early course of the outbreak in the locality, and is limited almost entirely to those who neglect to invoke the aid of the physician until the attack has become exceedingly grave. The general mortality among the attacked may vary between 20 and 80 per cent., according to the virulence or mildness of the type of the disease, the total average being nearly 50 per cent. If the patient is seen early and is promptly, judiciously, and *constantly* cared for, the danger of a fatal issue is usually not great.

Treatment.—Although the gross number of attacks of Asiatic cholera and the wide spread of pandemics of the disease among civilized nations have lessened considerably, thanks to better hygiene and improved methods of prevention, yet the percentage of deaths to attacks remains about the same now as it was many decades ago, and is not very materially lower under modern and civilized systems of therapeutics than it has been under antiquated and semi-civilized or barbarous modes of management. Knowledge of efficient methods of treatment of cholera has by no means kept pace with that of the etiology and prophylaxis of the disease. In the early stages of this disease the skilful physician is all powerful; in the latter stages he is almost

impotent. Hence the paramount advantage of prompt and judicious medical treatment.

TREATMENT IN THE PREMONITORY PERIOD.—During the prevalence of Asiatic cholera in a locality, every disturbance or derangement of the alimentary canal should be corrected without loss of time. Indigestion or abdominal distress should receive without any delay the careful attention of the physician, who should not fail to impress upon his clientèle the urgent necessity of scrupulous obedience to his instructions. The first thing to do is to remove any apparent cause of the disturbance; place the patient upon a lighter diet, fluids by preference; absolutely interdict any exercise which tends to overheat or fatigue; insist upon clothing during the day which will keep the trunk and extremities warm, and, during the night, which will prevent chilling of the abdomen and the legs. One article of clothing should consist of a broad flannel binder around the abdomen and loins next the skin, kept on day and night. The first appearance of diarrhœa should be the signal for active treatment. One or two stools during the twenty-four hours more than the usual number habitual to the individual when in health, or a single copious watery stool, should require the patient to be put to bed at once and kept recumbent, not only during the continuation of looseness of the bowels, but for a day or two after this condition has entirely disappeared. All solid food should be rigidly interdicted, and nothing but broth, bouillon, or whey, allowed to be eaten. In fact, an approach to abstinence is far more desirable than risk of overfeeding. The looseness of bowels or diarrhœa must be arrested as soon as possible, but in doing this it is much better to avoid powerful astringents and strong opiates if it can be done without them. In the choice of the remedy it should be borne in mind that the nature of the disturbance is that of a specific infection of the small intestine by the comma bacilli of Koch, associated with, and greatly favored by, a rather decided alkalinity of the intestinal fluids. The rational treatment would therefore seem to be the administration of some combination of acids, disinfectants, and sedatives. Of the acids which may be employed in proper doses are sulphuric, hydrochloric, lactic; of the intestinal disinfectants, naphthaline, salol, calomel, salicylate of bismuth; of the sedatives, paregoric, Hoffman's anodyne. Aromatic sulphuric acid and paregoric in proper doses may be given and repeated *p. r. n.* This may be alternated or not with naphthaline or salol, alone or in the same powder with salicylate of bismuth, or with naphthaline and calomel together. It will be found in the great majority of cases that this simple treatment will prove effective. Instead of the mineral acids, lactic acid is preferred by many. Dujardin-Beaumetz uses—

Ry. Lactic acid	prts.	10,
Syrup	"	20,
Tinct. of citron	"	2,
Water	"	1000.—M.

Sig. For the adult three teaspoonfuls, with or without 20 drops of paregoric added, at intervals of a half hour, or longer as the case may require.

As a drink instead of water, it is well to use an acid lemonade with a view to lessening the alkalinity or rendering acid, if possible, the reaction of the contents of the small intestine, in order to inhibit the growth therein of the specific microbe. Sulphuric, hydrochloric, or lactic acid—say, one part to the thousand of sterilized water, sweetened—may be employed for this purpose.

Should the diarrhœa persist or increase in severity in spite of the simple treatment above mentioned, recourse must be had without loss of time to more active medication. Stronger anodynes and decided astringents are called for. Chlorodyne may be used, or Lausedat's drops, as follows:

R \bar{y} . Tr. valerianæ æther. ℥c.
 Tr. opii ℥xx.
 Essentiæ menthæ piperit. gtt. v.
 Spts. ætheris comp. ℥c.—M.
 Sig. Five to eight drops for a child of six years.

Or something like the following may be tried:

R \bar{y} . Acid. tannici
 Plumbi acetat. āā gr. iij.
 Pulv. opii gr. ss.
 Oleoresinæ capsici gr. ij.—M.
 Ft. pil. No. XII.
 Sig. One pill every one to four hours, *p. r. n.*, at the age of six years.

On the principle of clearing the bowels of irritants and altering the secretions, some begin the treatment of this period with a large dose of calomel, followed in a few hours by castor oil combined with naphthaline.

TREATMENT OF THE PERIOD OF SEROUS DIARRHŒA OR SYSTEMIC INTOXICATION.—Although such early treatment as indicated above will, as a rule, prove effective in the prevention of full development of an attack, there are some cases which seem to be doomed, in spite of prompt and judicious attention, to advance into the period now under consideration. Moreover, it is usually not until this period that the physician is called. The conditions now to be contended with are those which have already been pointed out.

For the vomiting and thirst cracked ice and sinapisms to the epigastrium; for the coldness, envelop the whole person in hot flannel blankets, with bottles of hot water next the skin, and immersion in a hot bath for fifteen or twenty minutes at intervals of two to four hours; for the cramps, friction by rubbing with the palms of the hands: if the pain be violent it may be allayed by inhalations of ether; for the prostration and restlessness, cardiac stimulants and nervous sedatives; for the purging, chiefly intestinal antiseptics and correctives; for the loss of fluid, hypodermatic or intravascular injections of saline fluids; as against the special poison in the intestinal canal, irrigation of the colon with large injections of saline fluids.

Among the legion of remedies which have been tried and often been found wanting, the favorite East Indian compound called chlorodyne has been about as useful as any. Lausedat's drops, already mentioned, may take the place of chlorodyne. The remedies mentioned in treating of the prodromal period, especially the acids and antiseptics, may still be useful in the early part of the stage now under consideration. A powder which has been often used in former epidemics to combat coldness, prostration, and collapse has the following composition:

R \bar{y} . Bismuthi subnitrat. 5j.
 Plumbi acetat. gr. iij.
 Camphoræ gr. ij.
 Oleoresinæ capsici gr. j.—M.
 Divide in chart. No. XII.
 Sig. One every hour or two.

Macnamara, the great Anglo-East-Indian authority on cholera, says: "I think water, though urgently demanded by the patient, should be refused (cracked ice is recommended instead). I would restrict the opium to three grains; it is unwise to give more, although we are wellnigh certain that much of it has been vomited. . . . If the vomiting is very severe, a single dose of twenty grains (for the adult) of calomel will sometimes relieve this symptom. A mixture may be added, each dose of which contains two grains of acetate of lead and fifteen drops of dilute acetic acid, to be taken every second hour, and fifteen drops of dilute sulphuric acid in water every alternate hour, so that the patient should take a draught of first one mixture and then the other every hour. In this way the alkaline stools become acid, and perhaps destroy the cholera organism in the intestinal canal. However this may be, these acids seem to be beneficial in the treatment of cholera. . . . I believe that alcohol is positively harmful in any stage of cholera."

Unfortunately, in this stage of cholera medication by way of the stomach is always impeded, very often rendered almost useless, sometimes quite impossible of effecting an impression, by reason of the vomiting and the failure of absorption in the intestines. If the little that is not rejected by the stomach succeeds in reaching the intestine, it so often happens that none of it is absorbed; powerful drugs may lie and accumulate in the latter, to cause actual harm when the stage of reaction is ushered in, and with it restoration of the function of intestinal absorption. Neither can ordinary rectal injections of medicine be depended upon, for the same reason. The sluggishness, sometimes practical stagnation, of the little lymph still remaining in the tissues, after the continuous drain of copious watery evacuations from the bowels, usually lessens, often quite nullifies, the customary results of hypodermatic medication. When such a condition arises, as it unhappily too often does, what other resources has the physician left to him? There are still three which, used judiciously and skilfully, are powerful to restore marvellously—at least for a time, sometimes permanently—the suspended functions. I refer to intestinal, to hypodermatic, and to intravascular irrigation.

Enteroclysis, first introduced by the late Prof. Cantani of Naples during the former cholera epidemic in Italy as a means of treating all stages of the disease, consists essentially in irrigating the rectum, colon, and, if possible, also the small intestine, with large quantities of a warm, astringent, antiseptic, sedative fluid. The following is Cantani's formula for an adult:

R_y Boiled water or infusion of chamomile . . . 2 quarts.
 Tannin $1\frac{1}{2}$ to $2\frac{1}{2}$ drachms.
 Laudanum 30 to 50 drops.
 Powdered gum-arabic $1\frac{1}{2}$ ounces.

The temperature of this mixture when introduced should be sufficiently above the normal to aid in restoring heat to the body. Of course the quantity injected should vary according to the age of the patient and other circumstances in the judgment of the physician. The best time for administration is immediately after an evacuation.

Hypodermoclysis, also first introduced by Prof. Cantani as a means of treating especially the stages of serous diarrhœa and of algidity or collapse, consists essentially in the introduction hypodermatically of a large quantity of warm saline fluid for the purpose, primarily, of replacing the fluid lost through the intestinal drain; secondarily, of washing out from the blood and tissues much of the effete material and specific poison which have accumu-

lated in them. Cantani's formula for an adult consists of 2 quarts of boiled water, $2\frac{1}{2}$ ounces of pure sodium chloride and a drachm and a half of sodium carbonate. The quantity to be injected each time varies according to age, the apparent amount of fluid lost, and other circumstances. The amount for an adult is one to two and a half quarts. The temperature of the solution when injected should be $100\frac{2}{3}^{\circ}$ F., unless that of the rectum be very low, in which case it has been sometimes raised as high as $109\frac{2}{3}^{\circ}$ F. The most successful time for resort to hypodermoclysis is at the first indications of insufficiency of water in the body, such as Hippocratic countenance, wrinkling or discoloration of the skin, cramps, coldness, etc.

Intravascular injections of saline fluids, a procedure as old as the history of cholera in Europe, has had a renewed trial during the present visitation of the disease. Injection into veins and into arteries has been practised especially at Hamburg, and each method of procedure has its champions. Some variations in the constitution and proportions of the saline fluid used occur, but the following may be regarded as a standard: sodium bicarbonate 1 part, sodium chloride 6 parts, boiled water 1000 parts. The temperature of the fluid when injected varies according to circumstances from $100\frac{2}{3}^{\circ}$ F. to 104° F., more frequently the latter. The quantity administered has sometimes been very considerable, averaging for the adult one to two quarts. The injection may be repeated in a half hour to four hours, as the condition of the patient demands.

Of the relative advantages and disadvantages of the hypodermatic and intravascular irrigations, it may be said that the former is slower and usually more permanent in its action than is the latter. There may occur occasions, however, in the treatment of the algid period, when the matter of time will decide which method shall be tried first. It seems to me that it is mainly in rapidly-sinking cases in that period, that intravenous injection should be given the preference, to be followed at the second injection by hypodermoclysis. The hypodermoclysis has the further advantage of being far simpler of application. Only one skilful person is required for this operation; indeed, the attendants can readily be instructed to perform it very safely in the absence of the physician. On the contrary, the physician requires at least one skilled assistant to safely perform the intravascular injection. In all these operations strict antiseptic or aseptic precautions must be observed.

For enteroclysis there is needed a large fountain syringe with a long flexible tube with a cock, to which a moderately stiff but flexible terminal portion two or three feet long is attached. The tube, quite full of the fluid, must be passed up into the colon and worked along its interior as far as possible; the fluid should be let flow slowly, avoiding very sudden distention of the gut, and should be retained as long as possible.

For hypodermoclysis a fountain syringe with a long flexible tube, furnished with a cock, answers the purpose; with another shorter tube, one end attached to the cock, the other having a needle-pointed canula, a little longer, stronger, and with a somewhat wider calibre than the ordinary hypodermic needle. The tube and canula are first perfectly filled with the fluid, and then the canula is inserted well in between the skin and deep fascia of the flanks, buttocks, or interscapular region. The fluid should be made to flow slowly, allowing fifteen to twenty minutes for the introduction of one quart. The slight tumor should be made to disappear, as it will, by gentle kneading or massage.

For intravascular injections of saline fluids any good transfusion apparatus suffices.

Lavage of the stomach to stop vomiting is a most effective procedure, and sometimes succeeds in arresting this distressing symptom when nothing else will do it. Indeed, it would seem to be a very useful associate of enteroclysis, for it seems that to clear the stomach of the offending rice-water fluid is only second in importance to washing it out from the intestine. Boiled water holding in solution boracic acid has been satisfactorily used for this purpose.

TREATMENT IN THE PERIOD OF ALGIDITY OR COLLAPSE.—In this stage of the disease, where absorption is practically suspended, little is useful beyond enteroclysis and hypodermoclysis or intravascular injections of fluids, and efforts to communicate heat. The vast majority of cases in this stage die in spite of every effort of the physician, but there is certainly more success to be expected of this mode of treatment than of any other at present known.

TREATMENT IN THE PERIOD OF REACTION.—The treatment in this stage is essentially expectant and symptomatic. Each condition enumerated in the sections on Symptomatology and Etiology will suggest to the experienced the particular line to be followed. One of the most important things to avoid is pointed out forcibly by Macnamara, whom I can do no better than to quote in conclusion: "When reaction comes on, we must be careful not to fall into the error of over-feeding the patient under the mistaken idea of supporting his strength; he will not die of exhaustion if small quantities of milk and arrow root are administered frequently for two or three days, together with warm beef-tea enemas. But enteritis may certainly be induced if food beyond the simplest and smallest quantities be allowed. The patient requires rest and the most careful nursing after a severe illness like cholera."

Prevention.—Whilst the physician is often impotent in the treatment of cholera, in prevention he may be, if he will, all-powerful. It is not our purpose to discuss this subject from the standpoint of a state or community; we shall consider the matter solely from the side of the individual: First, what those ministering to the sick should do to prevent the spread of the disease; second, what the individual who may be exposed to the infection should do to safeguard himself from an attack of cholera.

1. *The Duties of those Attendant upon the Sick.*—I wish to say in the beginning that, whilst there is scarcely any infectious epidemic disease which is so capable as cholera of working great injury in various ways to the community, if the attendants upon the sick are ignorant or careless in applying the principles of prevention, yet there is no such disease which can so easily and certainly be limited to those attacked if only these principles be constantly and scrupulously applied. As I have said elsewhere, Asiatic cholera can be dwelt with and handled with absolute impunity if only the proper precautions be never once forgotten or neglected. There is, therefore, not the slightest danger in administering to the sick if carefulness be the rigid rule. It has already been pointed out that it is only the evacuations from the stomach and bowels of a person suffering an attack of Asiatic cholera that contain the original infection. To promptly and thoroughly disinfect these and everything soiled by them or containing them is to render the spread of the disease from the person attacked impossible. The evacuations should without any delay be treated in one of the following ways: *a*, water that is *boiling* should be poured upon them carefully, so as not to splash, in such amount that the volume of the water is four times that of the evacuations, or a strong solution of potash soap may be used in the same way; *b*, or fresh milk of lime (white wash), of twice the volume of the evacuation, should be poured upon the latter and the mixture gently stirred; *c*, or a similar quantity of a freshly-prepared solution (5 per cent. strong)

of chloride of lime may be used in the same way; *d*, or a similar volume of 5 per cent. solution of carbolic acid may be thus employed. Whichever one of these means be chosen, it is essential that the vessel be immediately covered from the flies and allowed to stand fifteen or twenty minutes before emptying; and it is also-essential that the disinfected evacuations be emptied into a pit in the earth, the bottom of which is covered with a layer of quicklime, and be covered immediately with another layer of the same material, care being taken that the location of this pit does not jeopardize water-courses, springs, or wells. Clothing or other textile fabrics soiled by the evacuations should be disinfected as soon as possible. They should be at first soaked in a disinfectant solution—say, a mixture of strong potash soap and carbolic acid of 5 per cent. strength—for an hour or more, and then boiled. It is better to burn bedding rather than attempt its disinfection. The floors of the sick-room should first be sprinkled with chloride of lime, and then mopped over with a cloth moistened in a chloride-of-lime solution. Any article of furniture which may have been contaminated should be carefully disinfected. Finally, it would be well to disinfect the room itself, after all is over, by means of sulphur fumes, 3 pounds to the 1000 cubic feet of space, for eight to ten hours. No one should be allowed in the sick-room except the necessary attendants, who under no consideration should eat or drink in this room. The patient should be fed from a set of dishes which should be disinfected immediately after use, and kept separate from those of the rest of the household; the remains of the patient's meal should be disinfected and destroyed. After handling the patient or anything that he has soiled, the attendants should immediately first disinfect and then carefully wash their hands: this thorough ablution should be performed invariably immediately before eating. After vomiting or an evacuation of the bowels the mouth and the parts around the anus should be wiped with a cloth wet with solution, 1:2000, of corrosive sublimate. If convalescence supervene, the patient should be kept isolated for a week, and the stools should be disinfected during that time. If death occur, the corpse should at once be enveloped in a sheet soaked with corrosive sublimate, 1:500, and cremated or buried without delay or funeral cortège. Finally, promptly notify health officials of every suspect or known case of cholera.

2. *Individual Precautions for the Exposed.*—No water or milk should be used or consumed, which could by any possibility be contaminated, unless recently boiled. No cold or uncooked food should be eaten which could possibly become contaminated. Such things as salads should be avoided. Unripe or over-ripe fruit should be eschewed. Alcoholic stimulants are pernicious. In fact, excesses of all kinds predispose to an attack. Regularity in eating, sleeping, exercise, and all other habits, contributes to safety. Keep all the bodily functions well regulated; avoid fatigue and chills. The use of a broad flannel waist-bandage next the skin day and night is beneficial in guarding against abdominal congestions. Quickly correct the slightest intestinal disorder.

DIPHThERIA.

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DIPHThERIA is an acute, contagious, and infectious disease, the most characteristic and constant feature of which is a pseudo-membranous exudate on, or a superficial necrosis of, a mucous membrane or some part of the skin which has been denuded of its epithelium. Although a comparatively recent disease in this country, it threatens to be the scourge of the large cities. Less than a century ago but few isolated and poorly-understood cases were seen, but the disease has spread very rapidly during the past fifty years, and in New York City alone the mortality from diphtheria and croup has exceeded fifty thousand in twenty-five years. And this number does not include many cases which were reported as deaths from pneumonia, nephritis, heart failure, etc., which were really complications of diphtheria.

There is no guide to the virulence of diphtheria. It is one of the most dreaded, one of the most fatal, and one of the most common diseases of childhood. At the onset it is impossible to say whether the disease will be mild or malignant. A case beginning with high fever and profound constitutional disturbance may go on to a rapid recovery; while, on the other hand, an apparently mild case will grow depressed and weak, and slowly die. Neither does the amount nor character of the exudate give any certain prognosis. Indeed, the clinical symptoms vary to such an extent that many mild cases are not even recognized unless some post-diphtheritic complication ensues; but, although these mild cases may be of small danger to the individual, they are all diphtheria and all equally contagious, and may be the origin of the most malignant ones.

Etiology.—It has been well recognized that certain cases of croupous inflammation are not true diphtheria. This list includes the chronic membranous exudates seen in certain forms of fibrinous bronchitis, cystitis, enteritis, etc., the acute superficial necrosis of the mucous membranes due to direct heat, as a scald, or an intense irritation from the application of ammonia. However, excluding these, there remain many doubtful cases; but modern bacteriological research seems to have solved this problem, and proven beyond much doubt that there are at least two forms of pseudo-membranous inflammation, the one a true diphtheria, due to the Klebs-Loeffler bacillus, and the other, which may include several varieties, a pseudo-diphtheria, due usually to a streptococcus.

True diphtheria is the product of the Klebs-Loeffler bacillus, either alone or associated with other bacteria, and it is primarily a local disease with many secondary manifestations, due to the absorption of the ptomaines or poisons which result from the growth of this micro-organism. The following observations seem to establish these propositions as fairly well proven:

1. This bacillus is present, usually in large numbers, in the false membrane

of all typical cases of infectious diphtheria, and is rarely or never found in other inflammations of the mucous membrane of the throat or in the healthy throat.

2. This bacillus is always found at the place of local infection, and never found in the blood or any of the internal organs, even though they may be the seat of marked secondary changes. On the contrary, streptococci and other bacteria may be found in the blood and internal organs.

3. Pure cultures of this bacillus when injected into the mucous membrane of susceptible animals produce a typical diphtheritic inflammation, even to paralyses and organic lesions.

4. Inoculation of animals with the toxalbumin of this bacillus produces the sepsis, the paralysis, the visceral lesions, and all the secondary constitutional symptoms of diphtheria, without the membrane.

5. Clinically, surface diphtheria, without participation on the part of the lymph-vessels, is apt to exhibit little or no fever; the disease does not run a typical course; one attack does not offer security against its recurrence in the future; and whenever the diphtheritic infecting agent finds a foothold on the body—as, for example, by inoculation—it always excites a local affection at the point of entrance; and from this local infection the general infection will develop, the extent and rapidity of which depend upon the anatomical relations of the affected parts, their characteristics, and their power of absorption.

The hypothesis that diphtheria is at first a general disease of the blood, with secondary manifestations on the mucous membranes, is hardly tenable in face of the foregoing facts. The chief arguments brought forward in support of this theory are its similarity to certain of the infectious diseases; its epidemic occurrence; the fact that constitutional symptoms may be present for hours and days before local symptoms are discovered; the marked susceptibility of children; the great disproportion often seen between the general symptoms and the apparently trifling local changes; the multiplicity of the localizations, and the fact that efforts to conquer the disease by destroying the pseudo-membrane with strong caustics have been for the most part without result. However, these observations simply prove that diphtheria may be a general infectious disease, but they do not explain how this infection takes place. Neither clinical observations nor post-mortem examinations have ever been able to present enough facts to settle this question; but, fortunately, modern bacteriological research, with inoculation experiments on living animals, has determined it very conclusively.

Besides true diphtheria, we frequently meet with an allied pseudo-membranous inflammation which cannot be distinguished from it clinically, except that it runs a milder course. Bacteriologically, however, the Klebs-Loeffler bacillus is always absent, and streptococci, and often other bacteria, are found in great abundance, not only in the exudate, but even in the blood and internal organs. The differential diagnosis is very important, as a knowledge of which disease we have to deal with modifies somewhat the treatment, and greatly the prognosis.

Not only do we have a croupous inflammation which is not a true diphtheria, but we can have a true diphtheria in which the membrane covers so little space that there is apparently no fibrinous exudate. This was clearly demonstrated by Jacobi in his article on "Follicular Amygdalitis;" and every observer must have seen cases in which an apparently catarrhal follicular amygdalitis quickly proved itself to be a diphtheritic one, or, after recovery, showed its true nature by a characteristic diphtheritic sequel—a paralysis of some muscle or group of muscles.

Accepting the microbic origin of diphtheria, we must still take into account the many conditions that materially modify the course of this affection, which is one of the most variable and uncertain of all the contagious diseases. It is doubtful if a normal mucous membrane can be infected by the bacillus, and it is certainly true that a lesion favors its development. This also applies to the toxalbumin of the bacterium, large amounts of which can be swallowed without danger by susceptible animals that have healthy and intact mucous membranes.

Age is ordinarily an important factor in influencing the occurrence of the disease; and, though it may occur at any time of life, it is essentially a disease of childhood.

Individual or family predisposition has some influence. It occurs by marked preference in connection with those diseases which produce lesions of the mucous membranes. Cold and dampness favor its occurrence, partly by their tendency to excite catarrhal affections and thus offer an opportunity for infection, and partly by the more favorable conditions for the growth of the bacillus which are present during such weather. All the windows and other sources of ventilation are shut, and the rooms, especially in tenements, where the disease is most common, are stifling and hot. Insanitary conditions undoubtedly favor the development of this germ.

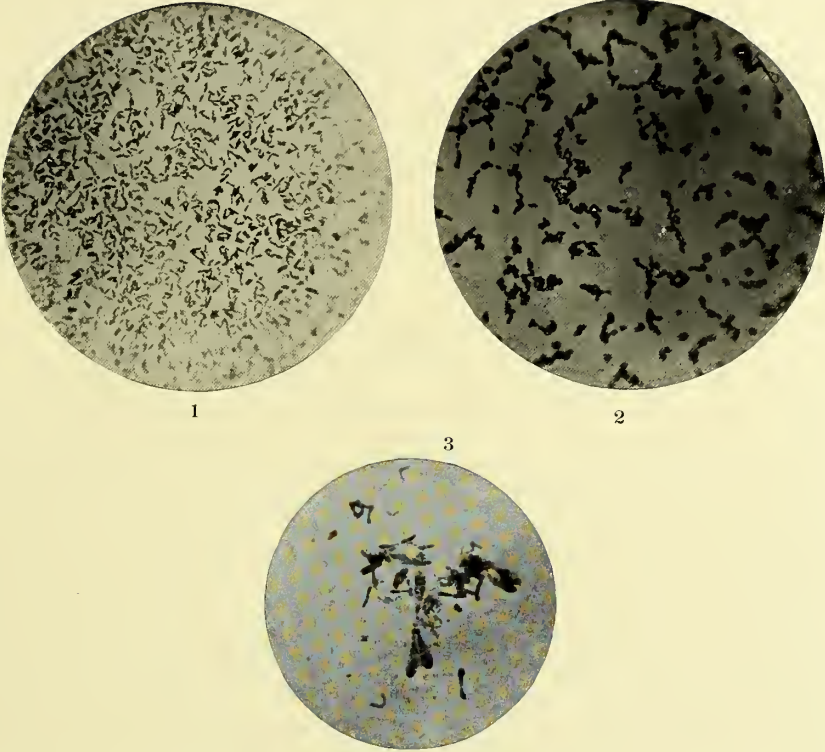
KLEBS-LOEFFLER BACILLUS.—In the membrane of true diphtheria this bacillus is always found, either alone or associated with other bacteria. It is rarely or never found in the blood or internal organs, although the streptococcus, which is often associated with it, may appear in the blood, the lymphatics, or the viscera. On the surface and the most superficial portions of the exudate the bacillus is found mixed with numerous other micro-organisms. In the middle or deeper portions the only organisms present are the Klebs-Loeffler bacilli, either alone or associated with streptococci. In the deeper layers there are only a few bacilli, and in the mucous membrane, as a rule, none.

These bacilli are "moderate-sized rods, usually slightly bent, averaging nearly as long as the tubercle bacilli, but twice as broad, and usually with rounded ends. According to the rapidity of growth, the soil, and other conditions, the form and size of the micro-organisms vary, and the differences are striking. The bacteria are sometimes enveloped in a more or less capacious membrane; sometimes the contents divide into a number of pieces, separated by transverse divisions; one end of the rod is frequently thickened like a club, or both ends may be clubbed, or one or both pointed. The bacilli are immobile and have no spores. The best staining agent is Loeffler's alkaline methyl-blue. Some forms stain uniformly, others in various irregular ways, the most common being the appearance of deeply-stained granules in a slightly-stained bacillus or of darkly-stained ends with a paler centre. The bacilli are very often in pairs, never in chains; they are semi-anaërobic, and thrive at a somewhat high temperature, 20° to 42° C."

"The Loeffler bacilli can be cultivated upon all the ordinary culture media, but grow most vigorously on a mixture of blood-serum and nutrient bouillon, as given by Loeffler. On this, solidified, the bacilli grow as large, round, elevated, grayish-white colonies, with the centre more opaque than the somewhat irregular periphery" (Park).

The most ready method of detecting this bacillus is to detach a small piece of membrane and place it for five minutes in a 2 per cent. solution of boracic acid, then to draw the piece of membrane along the surface of sterilized blood-serum in a test-tube, and maintain it at a temperature of 37°

PLATE IX.



4

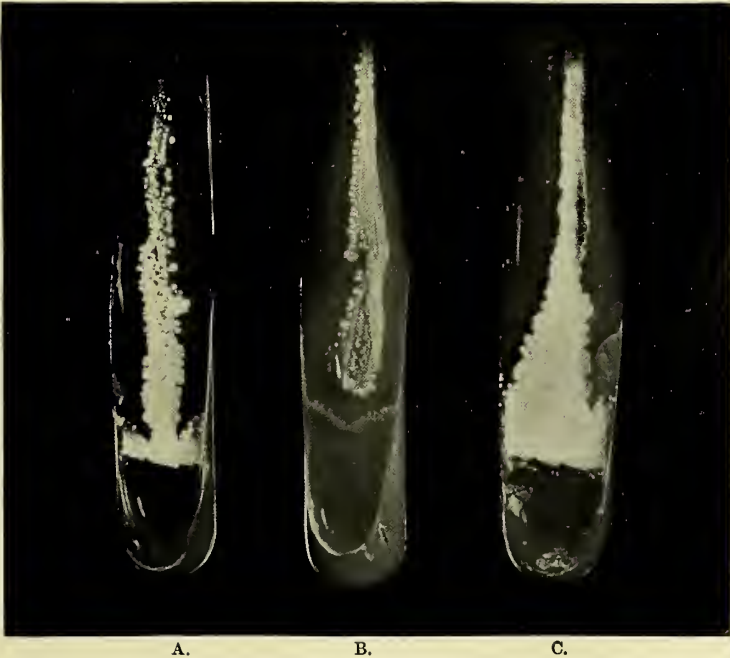


FIG. 1.—Loeffler bacilli. $\times 650$.

FIG. 2.—Pseudo-bacilli. $\times 650$.

FIG. 3.—Involution forms of the Loeffler bacillus. $\times 650$.

FIG. 4.—A. Pseudo-bacillus. B. True bacillus. C. Pseudo-bacillus.
(Natural size.)

From photographs taken by Dr. Henry Koplik, Carnegie Laboratory, New York.

C. for twelve to twenty-four hours. At the end of this time, if the bacilli are present, characteristic small white rounded colonies are visible along the track of inoculation. They can then be stained and examined. To get a pure culture a second or third preparation must be made. To overcome the difficulty of obtaining serum for the culture medium, Sakharof suggests the use of slices of hard-boiled eggs placed in sterilized test-tubes, and Johnston suggests the use of hard-boiled eggs from which a part of the shell has been removed with ordinary forceps, so that the shell-membrane can be peeled off and the inoculation made at that point. To guard the culture against contamination, the egg can be placed upside down in a common egg-cup, the interior of which has been sterilized by allowing a flame to enter it for a second or two.

The pseudo-diphtheria bacilli is a term applied to a group of micro-organisms which closely resemble the true diphtheria bacilli, both in appearance and in producing a pseudo-membrane, but they are without pathogenic properties in guinea-pigs, and they do not grow on gelatin at ordinary temperatures. However, for bedside diagnosis it is wiser to consider all cases as true diphtheria that give colonies of bacilli resembling the Klebs-Loeffler.

The ptomaine, or poison, produced by the diphtheria bacillus is of a proteid nature, precipitated by alcohol and soluble in water. When pure, it is a white amorphous mass and extremely poisonous. It is not at all, or but little, absorbed by healthy and intact mucous membranes; but when inoculated into a susceptible animal it produces all the symptoms of a diphtheria without the exudate.

Mode of Infection and Propagation.—There is no doubt that in the vast majority of cases the inoculation takes place through some lesion of the mucous membrane or of the skin. Therefore, it would be hard to over-estimate the value, as a prophylaxis, of attention to all lesions, no matter how slight, of the mucous membrane of the upper air-passages. Every catarrhal condition should receive prompt and efficient treatment, and bad teeth, accumulated secretions, or any other source of local irritation should be removed as soon as possible.

The germ is usually propagated through the surrounding air, and brought in contact with the mucous membrane during respiration. Less frequently the disease may be propagated by the direct deposition of diphtheritic matter by inoculation or through some article of food. It has been known to have been communicated from some of the domestic animals. The contagion may be spread by contact with the person or clothes of those suffering from the disease, and may also be spread by bed-clothes, furniture, and other articles in the sick-room. Too much care cannot be taken to prevent those surrounding the sick from spreading the disease, and there is no doubt that physicians themselves frequently carry the disease from one patient to another. This is clearly shown from the large number of cases which occur in their own families.

Incubation.—In experimental diphtheria the duration of the incubation period is short, varying from twelve hours to three days; but when diphtheria is contracted in the usual way—by inhaling air which contains the contagion—this period may be much longer, varying from one day up to twenty. However, in the latter case this only means the interval between exposure and the appearance of the disease, for there is no means of knowing exactly when the contagion entered the mucous membrane, and how long it had remained harmlessly upon it, waiting for the development of some lesion through which to infect it. It is obvious, therefore, that all observations based upon the interval between exposure and the appearance of the disease must be uncertain.

This period also depends not only upon the quality and quantity of the infecting material itself, but also upon the structure and texture of the tissues and their power of resistance—a power which is often greatly modified by streptococci and other bacteria which may be associated with the true diphtheria bacilli. When the Klebs-Loeffler bacilli are implanted upon a normal mucous membrane, they do not grow, but these associated streptococci produce an acute purulent discharge, with redness and swelling. Thus they prepare the lesion for infection by the Klebs-Loeffler bacilli.

Anatomical Changes.—The local pathological changes of this disease occur on a mucous membrane or some abraded portion of the skin. The changes found on the inflamed mucous membrane are as follows: The surface becomes hyperæmic and swollen, and presents the usual manifestations of a catarrhal inflammation. After a short time, usually a few hours, it is covered with a whitish or yellowish layer, which forms the pseudo-membrane so characteristic of the disease. This membrane may represent a fibrinous exudate which can be easily peeled from the surface beneath, or it may represent a true necrosis, so that the exudate is an integral part of the mucous membrane and cannot be separated from it. Many of its characteristics depend upon its anatomical position and the type of epithelium upon which it is located. It looks to the naked eye like coagulated fibrin, but under the microscope it is seen to consist of proliferated epithelial cells held together by a fibrinous network. In its physical and chemical properties it closely resembles fibrin. The surface beneath the exudate may show all grades of inflammation, from a mild catarrhal to an ulcerated one. The false membrane is found oftenest on the tonsils, uvula, soft palate, and back of the pharynx, the nasal passages, the larynx, and trachea; less commonly on the conjunctiva, at the border of the anus, or in the vagina; rarely in the bronchi as a primary affection, but not uncommonly as an extension of the same process from the larynx and trachea; and very rarely in the œsophagus, the intestinal tract, or the ear.

Besides these local pathological changes other organs of the body may become affected as the result of the absorption of the toxalbumin.

The adjacent lymph-nodes are swollen and inflamed, but they rarely become the seat of a suppurative inflammation; the surrounding tissues are infiltrated with serum containing scattered pus-cells.

The lungs show areas of intense congestion, with hæmorrhages into their tissue. They may exhibit œdema, broncho-pneumonia, catarrh, atelectasis, emphysema, ecchymoses, and large infarctions; and the bronchi may be lined with false membrane as far as the smaller branches. These changes, however, are mostly observed as complications of laryngeal diphtheria.

The pleura may be hyperæmic and inflamed, with hæmorrhages, and in many cases the pleural cavity will contain an excess of fluid.

The kidneys, in experimental cases, are moist and hyperæmic, and the adrenals are congested and may be hæmorrhagic. Fatty changes occur in the epithelium of the tubes and glomeruli, and hyaline alterations in the glomerular capillaries and in the smaller arteries. Hæmorrhages, parenchymatous and interstitial nephritis, are common lesions observed in the kidneys in albuminuric cases.

The spleen and the liver may be enlarged and congested, with hæmorrhages into the capsule and tissue. There may be present smaller or larger masses of necrotic cells, and in some cases there is a fatty degeneration, and occasionally, in protracted cases, a hyaline or a waxy one.

The heart may show in the substance of the muscle large and small hæmorrhages and ecchymoses. When death is due to asphyxia without

general poisoning of the whole organism, the muscular substance of the heart itself may be normal; but when there has been a general poisoning it has usually undergone a granular and fatty degeneration, and there may be other septic changes, as, for example, an endocarditis.

In both the parietal and visceral layers of the pericardium there may be small and large hæmorrhages and ecchymoses; there may be an excess of fluid in the pericardial cavity; and in rare cases there may be an emphysema of the pericardium as a consequence of the extension of a subpleural emphysema into the loose cellular tissue between the folds of the mediastinum.

The blood, as in most severe forms of septicæmia and poisoning, is but slightly coagulable, sticky, brown, or rather livid, and the blood-vessels contain a greatly increased number of leucocytes.

The mucous membrane of the intestinal tract and of the bladder may rarely become directly infected, and under such circumstances they present the characteristic pseudo-membrane and other changes which take place in the pharynx, etc. However, when secondary changes occur in consequence of general infection, cell-infiltration and hæmorrhages are the usual ones, and in one reported case such extensive hæmorrhage from the great omentum occurred that a considerable quantity of free blood had collected in the peritoneal cavity. The layers of the peritoneum may be injected and be the seat of ecchymoses, and the peritoneal cavity may contain an excess of serous fluid.

The fibres of the muscles show degenerative changes, and the thyroid may be congested and ecchymotic.

The earliest change in the brain and spinal cord is venous hyperæmia, both in the vascular linings and in the substance itself. Later in the disease come extravasations, with the subsequent softening of the surrounding tissue, and finally various degenerative changes. Extravasations into the substance of the spinal nerves have been seen, as well as granular degeneration of the nerves of the soft palate and other parts that have suffered from a diphtheritic paralysis.

Symptoms and Diagnosis.—The characteristic feature of the disease is the pseudo-membrane. There are cases of pseudo-membranous inflammation which are not diphtheria; but, excluding the chronic cases and those due to great heat, as a scald, and to the application of an intense irritant, like ammonia, it is often impossible to distinguish between the true and the false diphtheria, except by a bacteriological examination. The only positive test is the presence of the Klebs-Loeffler bacillus, either alone or associated with streptococci or other bacteria. In a certain proportion of cases it is very difficult to distinguish between the true and the pseudo-bacillus; and in all doubtful cases, at least for the present or until inoculation experiments can be made, it is wiser to consider them as true diphtheria. Clinically, cases of follicular amygdalitis are frequently diagnosticated as simple catarrhal or purulent inflammations, when they are really diphtheritic. All such cases should be isolated and treated in every respect as true diphtheria until the diagnosis is made certain either by a bacteriological examination or the appearance of new evidence which will show the true nature of the disease.

The diagnosis, even of a membranous inflammation, may be obscure from its location. It may be confined to the posterior nares, the larynx and trachea, or even the intestine, the bladder, or other positions where the local changes cannot be seen.

The constitutional symptoms which are the result of the poisoning due to the absorption of the toxalbumin produced by the specific bacilli vary greatly, and depend not only on the amount and rapidity of the absorption, but also

upon the susceptibility and condition of the patient. In simple and uncomplicated cases there is usually little or no fever. The symptoms may vary from this to evidences of the most profound poisoning. The temperature may be high and irregular, the pulse rapid or, in certain very fatal cases, abnormally slow. There is languor and loss of appetite, and an amount of prostration out of proportion to the fever and the local inflammation; the skin dry and hot; and, according to circumstances, typhoid symptoms may show themselves, or there may be delirium with great restlessness. Relapses are frequent, and one attack does not protect against a subsequent one.

The lymph-nodes which are in anatomical relation with the local process, as well as their surrounding tissues, may be swollen and tender, but they seldom undergo a suppurative change. The degree of enlargement and inflammation depends upon the amount of absorption, and of course this depends not only upon the character of the local process, but also upon its relations with the neighboring lymphatics.

The heart's action is usually rapid, and may be feeble, during an attack of diphtheria; and this condition often continues for some time after the disappearance of all local evidences of the disease. The pulse may be irregular both in force and rhythm. Another condition, usually appearing late in the disease, and often when the local process is apparently improving or has entirely cleared up, is for the feeble pulse to become progressively slower until the beats number less than forty, sometimes less than thirty, to the minute. These cases, which are nearly always fatal, together with those having the feeble, rapid pulse of profound sepsis and exhaustion, may be classed as examples of slow heart failure. But there is still another condition which usually appears after all the alarming symptoms are gone; that is, a sudden failure or paralysis of the heart. Endocarditis most frequently involving the mitral valve may occur, and is accompanied by fever, præcordial pain, attacks of syncope, a systolic murmur, and ante-mortem heart-clots, which may become free and enter the circulation, producing the usual phenomena. In most cases there is a rapid destruction of the red corpuscles of the blood, and a relative increase of the white corpuscles. Hence the anæmia which appears early and rapidly increases as the disease advances.

Albuminuria is a common complication, and appears usually on the third to sixth day, but may rarely appear as early as the first day or as late as the fifteenth. The amount of albumin varies greatly, from a slight cloudiness, on boiling, to complete consolidation. The urine usually appears normal, but it may be scanty and dark, and in rare cases dark-colored or smoky from the presence of blood. There may also be present in the sediment granular, hyaline, epithelial, and blood casts. The duration of the renal complication varies from a day or two to a week or two, but it may occasionally become chronic. It is seldom attended with œdema, but vomiting and other uræmic symptoms are not so rare. It is impossible to distinguish between the albuminuria of true and of false diphtheria, but in diphtheria there are some characteristics which distinguish it from the same complication of scarlet fever.

The tonsils are the most frequent location of the disease, and when confined to them it runs a mild course, because they have little or no connection with the lymphatic system, and they do not contain a large number of blood-vessels. The chief difficulty in diagnosis is to distinguish between a simple follicular amygdalitis and a diphtheritic one. The secretion from a catarrhal amygdalitis may cover the tonsils with a coat which closely resembles pseudo-membrane, but it can be easily washed away with a syringe, and in most cases a careful examination will show its true character.

The pharynx, soft palate, and mouth may be involved; and here it is a more serious condition than when confined to the tonsils. The lymph-vessels are very numerous: those of the uvula connect with the deep facial glands; of the tongue, with the deep cervical and the submaxillary glands; and of the floor of the mouth, with the submaxillary glands. The differential diagnosis lies between true diphtheria and false diphtheria, exudates as the result of an intense heat or irritation, ulcerative and gangrenous stomatitis, or occasionally herpes and aphthæ. The main differential symptoms pointing to diphtheria are, besides the history, the characteristic pseudo-membrane, the thin, brownish, acrid discharge, the sweetish and musty fætor, the glandular swellings, the tendency to hæmorrhages, the slight fever and marked prostration, the albuminuria, and the sequel of paralysis. In doubtful cases the only positive demonstration is the presence of the pathognomonic bacilli.

In the nares diphtheria is very serious on account of the abundant lymph- and blood-supply, and the consequent increased facilities for absorption of the poison, and on account of the conformation of the nasal passages, which interferes with their thorough drainage when swollen and inflamed, and which makes thorough local treatment very difficult. The greater supply of lymph-vessels is in the inferior portion of the nasal cavities, and these vessels connect with the deep facial and posterior submaxillary glands. It is often very difficult, and may be impossible, to see the pseudo-membrane in the posterior nasal cavities in children. Theoretically, it is very simple to use a rhinoscope, but practically it is quite another matter, and it is often impossible and usually impracticable, even in a tractable child. The symptoms which help to a diagnosis are the thin, acrid discharge more or less stained with blood, the evidence of nasal obstruction, the enlarged cervical glands, the bad odor to the breath, the tendency to hæmorrhage, and the frequent signs of general poisoning.

When the epiglottis, larynx, and trachea are involved, the main danger comes from the mechanical obstruction to respiration and the extension of the disease to the bronchi. Constitutional symptoms are usually absent, partly on account of the protection afforded by the very numerous mucous glands, and partly on account of the absence of lymphatic glands and the scant supply of lymphatic vessels. These vessels connect with the bronchial glands. After death from laryngeal diphtheria these glands are found more or less enlarged.

The diagnosis by means of the laryngoscope would be very valuable if it were practical. In the vast majority of cases it is not only impossible, but it is unnecessary and cruel. There is undoubtedly a membranous laryngitis which is not diphtheria, but the differential diagnosis cannot be made either from the symptoms or the character of the membrane. It can only be made by a bacteriological examination, which will show the presence or the absence of the Klebs-Loeffler bacillus.

The differential diagnosis lies between a membranous laryngitis and a catarrhal or a spasmodic one; and while this is not usually so very difficult, certain cases will present phenomena which keep the diagnosis obscure, unless the membrane is actually seen through the laryngoscope or is coughed up. Again, cases which, in the beginning, are catarrhal and run a typical course, may later become infected and run the usual course of a membranous inflammation. Again, confusion may be caused by those rather rare cases in which the membranous inflammation begins below and ascends to the larynx.

In the uncomplicated cases of membranous laryngitis, excluding the ascending ones, there is little or no fever; the onset of the disease is gradual, and it grows progressively worse; there is hoarseness, and after a time complete

aphonia; the stenosis is, at first, slight and only on inspiration, but after a while, usually two to four days, the stenosis becomes extreme, and attends both inspiration and expiration; the respiration and the cough, which in the beginning may be noisy and croupy, gradually become more husky and suppressed. Spasmodic attacks may occur in connection with the disease, but this is not a prominent feature of its clinical history.

The cases of ascending diphtheria of the trachea and larynx are very fatal, and, fortunately, uncommon. There are no constitutional symptoms, and the only evidence of sickness which can be detected is a slight bronchial or tracheal catarrh, until the process reaches the subglottic division of the larynx or the chink of the glottis, when laryngeal symptoms are seen, and stenosis appears and increases so rapidly that the patient becomes cyanotic within an hour or two, and soon dies unless immediate relief is given by intubation or tracheotomy. Even after operative interference the patient, in most cases, dies from extension of the disease to the bronchi, and usually within two days.

In doubtful cases the appearance of membrane in other locations, or the existence of an epidemic of diphtheria, favors the diagnosis of a membranous laryngitis. If with this the temperature is low, not high; the stenosis increases progressively, not spasmodically; the onset is gradual, not sudden; the laryngeal symptoms are of long, not short duration,—the diagnosis of membranous laryngitis is very clear.

There are numerous evidences of stenosis of the larynx besides the noisy respiration, as this latter symptom may be present in many other conditions. The most characteristic features of laryngeal obstruction are the deep recessions of the soft parts of the chest in inspiration, the blue or leaden hue of the skin and mucous membranes, the aphonia, the restlessness, and the abnormal frequency of the respirations; but none of these symptoms are constant. The soft parts of the chest-walls may project and make the chest barrel-shaped if the obstruction is greater on expiration; the skin and mucous membranes may appear blue if the stenosis increases rapidly, but this color becomes a leaden white if the obstruction is of slow progress; the voice may be clear in subglottic cases; and in advanced cases the restlessness is supplanted by a condition of stupor from carbon-dioxide poisoning. The only constant and reliable guide as to the presence and the amount of laryngeal obstruction is obtained by auscultation of the chest and listening to the respiratory sound. This gives an accurate guide as to the amount of air entering the lungs.

The other diseases which should be considered in making a diagnosis are abscess of or about the larynx, tumors of the larynx, retropharyngeal abscess, certain cases of naso-pharyngeal obstruction, foreign bodies in the air-passages, etc.; but the diagnosis should not be difficult if a careful examination is made.

In the bronchi a membranous inflammation is rarely or never primary, but is secondary to a similar one in the larynx or trachea. It may extend to the finer bronchial tubes, or even into the air-cells themselves, and result in a bronchopneumonia, with pulmonary collapse or emphysema. Its symptoms are, in a case of laryngeal diphtheria, a sudden rise of temperature—often very high—rapid respiration, marked dyspnoea, and cyanosis; and, although the physical signs in the chest are often obscure and masked by the laryngeal disease and pulmonary complications, there is less air entering the lung on the affected side, and the respiratory sound is dry and “boardy.”

Diphtheria of the conjunctiva, the ear, the intestinal tract, the genito-urinary organs, abraded portions of the skin, and wounds has occurred, usually

as a secondary process, but occasionally as a primary infection. The symptoms are those of an ordinary inflammation in those parts, to which are added the pseudo-membrane and other characteristics of this disease.

Diphtheria may, of course, complicate any disease, but the most frequent association is with scarlet fever, measles, and those diseases which present a catarrhal condition of the mucous membranes, and thus favor a fresh infection.

The skin eruptions which occur in diphtheria are septic manifestations, and may be of three kinds. The mildest and most transient closely resembles a scarlet-fever rash, but disappears more rapidly and does not desquamate. The second type is a purpura hæmorrhagica, and is usually associated with septic and grave forms of the disease. The last type, also seen in scarlet fever, usually follows a purulent septic infection, and occurs in cases which have a high mortality. There is an increase of temperature and the invasion is gradual. The eruption appears as red or dark-pink blotches, with sharply-defined margins. The color fades on pressure with the finger, but quickly returns. It appears first over the prominent bony points, such as the ankles, finger-joints, elbows, outer sides of the feet, etc., but always has a tendency to become general. Its disappearance is followed by a profuse desquamation, and usually this is quickly followed by a return of the eruption.

Sequelæ.—Besides the chronic catarrh which is left at the site of the pseudo-membranous inflammation, and the anæmia, the most frequent and characteristic sequel of diphtheria is paralysis, which develops from one to five weeks after all evidence of the acute disease has gone, though it may make its appearance during the course of the primary affection. It is a true multiple, peripheral neuritis, and resembles very closely, both clinically and pathologically, the neuritis of alcohol, lead, and other poisons. The duration of the paralysis usually varies from two to six weeks; it may last several months, and in exceptional cases has persisted for years. It is more frequent in adults than in children, and the severity of the paralysis or the probability of its appearance. Recovery usually takes place, and, while the location and the order of involvement differ greatly, the course is usually as follows: The soft palate and pharyngeal muscles, giving a nasal tone to the voice and a tendency to regurgitation of food through the nose during deglutition; the muscles of the tongue, lips, and face; the ocular muscles, as shown by strabismus and disturbances of vision; the lower extremities; the upper extremities; the larynx, recognized by modifications in the character of the voice or by obstruction, usually on inspiration; the muscles of the neck, with inability to control the position of the head; the muscles of the trunk, with loss of power over the body; the intercostal muscles, the diaphragm and other muscles of respiration, with interference with their function; the heart, usually fatal, but may not be; the walls and the sphincter of the intestines or bladder. There has also been observed paralysis of the special senses, giving temporary amaurosis, deafness, and impairment of taste and smell.

The paralysis of diphtheria may be divided into two classes; first, a true multiple neuritis, with loss of tendon reflexes as the result of poisoning by the toxalbumin; and second, other types of paralysis, as a result of hæmorrhages or degenerative changes in the brain or spinal cord. The first type occurs only in true diphtheria; the second may occur in true or false diphtheria or as a result of many other septic conditions.

Prognosis.—The prognosis is always better when the Klebs-Loeffler bacillus is absent. In 159 observations on cases of pseudo-membranous inflammation

made by Park at the Willard Parker Hospital of New York, the Loeffler bacillus was found in 54 cases, and in the other cases streptococci were the most abundant bacteria, and often the only ones. The mortality in true diphtheria was $46\frac{1}{2}$ per cent.; in pseudo-diphtheria, $5\frac{2}{3}$ per cent.; intubation in diphtheria, $71\frac{1}{2}$ per cent.; intubation in pseudo-diphtheria, $28\frac{1}{2}$ per cent.; adults in diphtheria, 36 per cent.; adults in pseudo-diphtheria, 2 per cent.

The prognosis varies, not only according to the age and condition of the patient, to the symptoms, and to the anatomical location of the disease, but also according to the character of the prevailing epidemic. The danger is greater the larger the surface involved and the more the exudate approaches a septic or gangrenous type, as shown by broken-down masses of exudation, the sweetish foul odor from the mouth, the yellowish or brownish secretion from the mouth and nose, which is both fetid and acrid, and the swelling and tenderness of the lymphatic nodes and the surrounding cellular tissue. However, the prognosis must always be a guarded one, since the subsequent course of the disease can never be predicted; and even after it has apparently terminated in recovery a relapse may take place, the infection may extend to the larynx or nose, or sudden death may result from paralysis of the heart. Another class of cases result in death, after all local manifestations of the disease have disappeared, from a slow exhaustion. Such a condition might be called diphtheritic marasmus, the chief characteristic of which is the distaste for all food and the progressive and extreme emaciation.

The prognosis in nasal cases is more serious, for reasons already given, while in laryngeal cases the prognosis is very grave from the great danger of asphyxia; and, even if this be overcome, from the ease and frequency with which the membranous inflammation extends into the bronchi.

Unfavorable prognostic signs are pallor, prostration, vomiting, hæmorrhages, marked weakness of the pulse, with excessive rapidity or slowness, fetor, purpura hæmorrhagica and septic blotches on the skin, persistent high fever, restlessness, delirium, and anorexia. The importance of albuminuria depends upon its character and the gravity of the symptoms which are associated with it. Diphtheritic paralysis usually ends in recovery, and is dangerous only when it involves the heart or the muscles of respiration or deglutition; and even in these cases its danger depends upon its degree.

Prophylaxis.—The first requisite, after the appearance of the disease, is complete isolation of the patient, either in a hospital devoted to contagious diseases or in a separate room in the house, preferably on the top floor, and containing as little furniture as possible. Separate dishes and other utensils should be kept for the sick-room, and everything that it is necessary to return to other parts of the house should be thoroughly disinfected before it leaves the room. All discharges should be received in vessels containing a strong solution of copperas or corrosive sublimate. The clothing, towels, etc. should be put in a solution of sulphate of zinc (4 ounces) and common salt (2 ounces) in boiling water (1 gallon). Water-closets, privies, etc. should be liberally treated with copperas solution ($1\frac{1}{2}$ pounds to the gallon). During the continuance of the disease it is of great service to keep the room filled with some antiseptic vapor, as carbolic acid, eucalyptus, or turpentine; but I have found that most good in preventing the spread of the disease is obtained by subliming fifteen to thirty grains of calomel in the room every hour. After recovery the patient should be thoroughly cleansed and disinfected, and dressed in clothes that have not been exposed to infection. In any event, as much as possible of the exposed clothing, furniture, etc. should be destroyed, and the rest thoroughly disinfected, either by the methods previously described or by naphtha or super-

heated steam. The walls, bed, and furniture should be washed with a strong solution of corrosive sublimate, and then, after closing the room tightly, sulphur should be burned in it in the presence of an excess of moisture—about three pounds of sulphur to every thousand cubic feet of air-space. After this it is well to advise that four to eight ounces of calomel be sublimed. Other members of the family should be kept from school and church; they should be removed to a different house if possible, away from the infection, and their naso-pharyngeal cavities and teeth should be kept clean by means of antiseptic washes, sprays, and gargles. At all times, and especially during an epidemic or after exposure to it, the mucous membrane of the respiratory tract should be kept in as healthy a condition as possible by keeping it clean and free of lesions.

The physician should protect his clothing as much as possible on entering the sick-room by a linen gown, and before seeing another patient, especially a child, all parts exposed to the infection should be thoroughly aired; or, better still, he should disinfect himself and put on fresh clothes, leaving the discarded ones exposed to the open air or to the fumes of subliming mercury.

One of the chief causes of the spread of diphtheria in New York City is the laxness, and almost criminal carelessness, of the authorities in our dispensaries for the poor. It is almost a daily occurrence in the large dispensaries for a contagious case to be packed in a small, hot room with a number of other children, most of them ill and in good condition to contract the infection.

Treatment.—There is no disease in which a greater variety of treatment has been recommended—from the expectant, which lets the patient absolutely alone, to the active treatment, which requires him to be disturbed every few minutes. It is impossible to lay down any routine plan: we have no specific for the disease, and each case should be treated on general principles and according to its individual indications. The general condition and strength of the patient should be improved as much as possible. There should be plenty of sunlight and fresh air in the sick-room, which should be kept at a uniform temperature of about 70° F. The clothes and bed-linen should be kept clean and pure by frequent changing. The skin should be kept in good condition, and special care should be taken of the digestion and nourishment. Great stress should be given in advising the recumbent position and avoiding all exertion, but, of course, this is often impossible in children.

INTERNAL TREATMENT.—Alcohol and food are of the greatest value, and too much stress cannot be laid on the importance of their proper use. The diet should, as a rule, be a liquid one, and consist of such food as is easily digested. Cows' milk, pure and fresh, is undoubtedly the best, but to aid digestion or to prevent souring and other fermentative changes it may be peptonized, or lime-water or an antiseptic may be added to it. To give variety to the diet or to meet special indications other wholesome and nourishing articles may be included, as beef juice, eggs, etc. The food should always be given at regular intervals, about once every three or four hours, and in definite quantities. It is always harmful to compel a child to take more food than it can digest, and any drug which interferes with the proper digestion and assimilation of the food is positively harmful, and its use should be avoided.

Alcohol, as brandy, whiskey, champagne, wine, or in some other form, should be given rather freely from the beginning, and there is more danger from giving too little than too much. A three-year-old child can take from one to ten ounces of whiskey in twenty-four hours, and in bad septic cases this amount may be greatly increased with advantage. Other valuable stimulants are carbonate of ammonium, camphor, musk, strychnia, digitalis, and the large

number of heart stimulants and tonics; but alcohol, in one of its many forms, is by far the best and safest.

The remedies which are given internally in the treatment of diphtheria make a long list, but most of them are of doubtful value, and many of them interfere with the digestion or do positive harm in other ways. Tincture of the chloride of iron is the most popular one. Locally it is a powerful astringent and antiseptic, but internally it seems to me that the theoretical benefit which it produces is, in many cases, more than counterbalanced by the digestive disturbances which follow its use.

Chlorate of potassium has an excellent effect in healing lesions of the mucous membranes, but internally, especially in large doses, it is positively dangerous, not only by its irritating effects on the stomach and intestines, but also by its dangerous action on the kidneys and heart.

The mercurials, especially the corrosive and the mild chloride, are undoubtedly valuable, but most of the good resulting from their use is obtained from their local effect on the pharynx, and their local effect in the digestive tract by preventing fermentation. The corrosive sublimate should be used in large and frequent doses, and always well diluted.

Turpentine, chloride of ammonium, iodide of potassium, antimony, the salicylates, bromine, benzoate of sodium, balsam of copaiba, cubeb, quinine, pilocarpine, and many other drugs are enthusiastically advised by different writers; but in the light of recent knowledge of this disease it is difficult to understand how any benefit could be obtained by their internal administration.

High fever should be reduced by sponging and baths, and the antipyretic drugs, antipyrine, acetanilid, phenacetin, etc., should be avoided, because they all increase the depression of the weak and degenerated heart. The bath, if used, should not be cold, but begun at 95° F. and gradually reduced to 80°, or even 70° in bad septic cases. Stimulants internally, hot applications to the extremities, and a warm sponge-bath are valuable in overcoming any bad effects of an over-cold bath. However, it is seldom wise to reduce the temperature of the bath below 70° F., and the best antipyretic effects are obtained in this manner. The patient should remain in the bath until the temperature, taken in the rectum, begins to fall, when he should be immediately removed and put to bed. In laryngeal cases, and in cases with enlarged and tender lymphatic glands, cold applications, and even the ice-bag, often seem to be of benefit to the local process.

Exhaustion, reflex vomiting, collapse, diarrhœa, hæmorrhages, and other complications should be treated symptomatically and promptly; but their appearance can often be prevented, and every effort should be made to attain this end. For exhaustion and collapse alcohol in large doses, both by mouth and under the skin, is the best remedy, but digitalis, nitro-glycerin, strychnine, camphor, and musk are useful. In the rapid heart failure of diphtheria, with an irregular and fluttering pulse, nothing is equal to a moderately large dose of morphine, given hypodermatically. It is a powerful stimulant, and quiets and steadies the heart. For the reflex vomiting there is nothing more satisfactory than the oil of wormwood, given as follows:

R_y. Olei absinthii gtt. j to ij.
Sodii bicarbonatis ʒj.
Aquæ menthæ piperitæ ad fʒiv.—M.

Sig. One teaspoonful for a child three years old, every half hour until the vomiting ceases. Shake well before using.

When the vomiting is due to uræmia or to irritation of the stomach other appropriate measures should be taken. For the diarrhœa, when due to local irritation in the bowels, give an active cathartic, by preference calomel or castor oil, to remove from the digestive tract the cause of the irritation, and follow this by an antiseptic to prevent further fermentation. The following answers very well:

R̄. Hydrargyri chloridi corrosivi gr. j.
 Bismuthi subnitrat̄is ℥iv.
 Aquæ anisi f℥iv.—M.

Sig. One teaspoonful in water every two hours until the discharges are black and lose their fetor. Shake well.

In severe hæmorrhages, especially from the nose, it may be necessary to apply local astringents or even to plug the nares with cotton. However, this should be avoided when possible, and many cases, being caused by an irregular and weak heart or a passive congestion from a weak right ventricle, can be stopped by the use of alcohol, digitalis, or nitro-glycerin, according to the indications.

LOCAL TREATMENT.—It must be acknowledged that the best and most satisfactory results in diphtheria are obtained by local treatment. The chief points to be considered in deciding upon a plan of treatment are—

1. *The most convenient method of applying the medication*—by spray, irrigation, insufflation, gargle, inhalation in the form of vapor, or by direct application with a swab. This will vary according to the medication employed and the location of the disease. For naso-pharyngeal cases the most satisfactory and thorough method is by irrigation with a fountain syringe. Through the nostrils the whole naso-pharyngeal cavity can be most thoroughly cleansed, and with less difficulty than by any other method. The child should be kept in a horizontal position when possible, and a rubber sheet arranged to catch the discharge. At each irrigation it is necessary to use enough of the solution to thoroughly clean the naso-pharynx—about one pint. This should be done every two hours, and in all cases often enough to thoroughly clean the diseased surface and bring the germicide in direct contact with it. In adults it is very satisfactory to use the irrigation through the mouth. In children this is often impracticable, but, when necessary, pass the nozzle of the syringe back between the teeth and cheek, so that the stream will enter the pharynx behind the last molar tooth. If the child be intractable and exhaust himself to a dangerous degree by fighting against the treatment, it may become necessary to clean the surface by giving internally plenty of water, either alone or with a weak antiseptic or a mild alkali, and applying the germicide by inhalation in the form of vapor, either by the sublimation of fifteen to forty grains of calomel every hour or two, or by keeping the air of the room saturated with steam which is impregnated with turpentine or some of the volatile antiseptics. The following is an excellent combination:

R̄. Acidi carbolici f℥j.
 Olei eucalypti f℥ij.
 Spts. terebinthinæ f℥viiij.—M.

Sig. Add a tablespoonful every half hour to about a quart of water, which is kept simmering over a flame.

In laryngeal or bronchial cases, although an application may be made

directly to the larynx with a swab, the only satisfactory method is by means of the inhalation of a medicated vapor.

The spray, while of value, usually does not go beyond the oral cavity, and seldom or never reaches the posterior pharynx. Through the nose it does better service. The swab, except in very careful and experienced hands, is liable to be too harsh and tear off membrane, thus opening up fresh avenues for infection; and in the grave cases, the nasal ones, it is almost useless. The use of the gargle is limited to adults and older children, it is not thorough, and it tires the patient very quickly.

2. *The medication to be employed.* There are two indications to be met: (a) the clearing away of débris and dead tissue, which may be the cause of much fetor and secondary septic complications, and which may also prevent the germicide from reaching the bacilli; and (b) the destruction of the living bacilli and other bacteria which are producing the disease. A third indication would be to neutralize or to destroy any of the unabsorbed toxalbumin which may be present. With our present knowledge of the properties of this poison it would be difficult to decide upon any practical rules, but we may be sure that its mechanical removal by irrigation is of value. We know that it is taken up very slowly from the infected tissues, often giving symptoms of fresh absorption after all the bacilli have disappeared; therefore the importance of keeping the surface of the mucous membrane clean after all evidence of the disease has gone.

(a) The most efficient drug for the removal of broken-down membrane, dead tissue, pus, and other débris is the peroxide of hydrogen, although it has apparently no destructive effect on the living bacilli. For this purpose it is certainly superior to any other means, although there are some preparations which are of great value as adjuncts—*e. g.* a saturated solution of borax in hot water, and the solvents, like pepsin, trypsin, and papayotin. The ordinary fifteen-volume solution of peroxide should be used, either in full strength or diluted with lime-water, which removes some of its acrid and irritating qualities without impairing its efficiency. It should be used freely, and in most cases a mixture of one part of the ordinary fifteen-volume solution with two or three parts of lime-water is effective. The best method to apply it is by irrigation with a fountain syringe, using about half a pint each time, and often enough to keep the diseased surfaces clean. There are several objections to its use. One is the difficulty of obtaining a fresh and active solution. This objection has been, in a great measure, overcome by Squibb of Brooklyn, who has made it possible to freshly prepare this solution at the time of use. A serious objection to Squibb's method is the long time required to prepare the solution. However, it is always well to test the activity of the solution before depending upon it. Another objection, and an important one, is its irritating effect upon the mucous membrane. It causes pain, and, as a result, objections to its use on the part of the patient; it also produces fresh lesions in the healthy mucous membrane, thus offering new places for infection. In my early experience with the drug, these objections and the greatly increased number of cases in which the diphtheritic process extended to the buccal mucous membrane, the gums, the tongue, and lips, seemed to make its use of very doubtful value, and probably harmful. However, these faults can be obviated in a great degree by diluting the solution with an alkaline water, and, after its use, by irrigating the same surface with a saturated solution of borax in hot water. If it is desirable to use the solution of peroxide without diluting it, neutralize the excess of acid with an alkali.

(b) To destroy the bacilli almost every caustic, astringent, digestive ferment, essential oil, and germicide has been lauded, and brilliant results claimed for each. Unfortunately, most of these reports are not based upon enough observations to be of much value; and it is apparently not recognized that nearly every case of tonsillar, most cases of pharyngeal, and many cases of naso-pharyngeal diphtheria recover under any kind of treatment.

Of all the germicides, the mercurials seem to have the most destructive effect on the Klebs-Loeffler bacillus, and carbolic acid, either alone or combined with eucalyptus and turpentine, on the streptococci and other bacteria which produce the false diphtheria. As it is often so difficult to distinguish between them—and, in fact, both forms are so frequently combined—it is better to use locally both the carbolic acid and some mercurial preparation. Therefore, always keep the room moderately filled with steam that is impregnated with the mixture of carbolic acid, eucalyptus, and turpentine. In naso-pharyngeal cases, after the thorough cleansing of the surface with the peroxide and the borax solution, use in the cavity a solution of bichloride of mercury, 1:1000, either by irrigation, with a swab, or by spray. No metallic utensils should come in contact with the mercury solution, as it corrodes them. If, for any reason, it is impossible to use the irrigation or spray, the local effect of the mercury may be obtained by subliming the mild chloride and allowing the child to inhale the fumes.

In laryngeal cases dependence must be placed upon inhalation, as it is impracticable and dangerous to use the laryngeal applicator. The inhalation most destructive to the Klebs-Loeffler bacillus is the fumes obtained by subliming calomel. The child should be well wrapped up, so that only the face is free, thus exposing the least possible surface of the skin to the action of the mercury. It should then be placed in an ordinary croup-tent, and the calomel sublimed in such a manner as to fill it with the fumes. The best apparatus for this purpose is the ordinary steam-spray, in which the boiler is replaced by a strip of tin upon which the calomel is put. Another good arrangement is to put a small alcohol lamp in the bottom of an ordinary chamber, and cover it with a pie-pan or strip of metal to hold the powder. The same end may be attained with a hot stove-lid, a shovel of red-hot coals, and in other ways. According to circumstances, fifteen to forty grains of calomel should be burned in this manner every one, two, or three hours. It is not necessary to wake the child for treatment, and if the smoke causes much coughing and irritation, subliming it less rapidly by lowering the flame of the lamp. It usually takes about ten minutes to sublime fifteen grains, and if care be taken to obtain pure calomel, or, better yet, calomel which has been recently sublimed and recondensed, the irritation from the fumes is usually very slight. This treatment does good not only by its local effect in the larynx, but by keeping the bronchi protected, and thus preventing the most common and fatal complication of laryngeal diphtheria—the extension of the disease to the bronchi. This treatment, which was first suggested by Corbin of Brooklyn, is not only of great value after operative interference, by preventing the extension of the disease to the bronchi, but its early use will in many cases obviate the necessity of an intubation or a tracheotomy. Besides this, it keeps the sick-room disinfected and helps to prevent the spread of the disease. The attendant should be cautioned to inhale the vapor as little as possible, as it is surprising how frequently the nurse becomes salivated and how seldom the patient is at all affected. However, this treatment seems to have a depressing effect on some patients, although there are seldom any other evidences of mercurialization; but it should be remembered that in infants and young children mercury is not liable to produce salivation

as in adults. Its effects are shown rather by marked anæmia and depression, with signs of irritation of the intestines and the kidneys.

The operative treatment of diphtheria will be considered elsewhere, but the following suggestion may be of value in overcoming one of the most serious complications that arises—namely, loose membrane in the trachea or bronchi. Its removal by aspiration, by tubes of large calibre, and by numerous kinds of forceps has been attempted, but with little or no satisfaction. The most successful method in my own practice is to insert a small laryngeal applicator, the cotton on which is covered with a very sticky substance like Canada balsam. Upon its withdrawal more or less of the membrane remains adherent to it, and after several trials and in many cases the loose membrane is all brought out.

ANTITOXIN.—In discussing the value of any treatment for diphtheria it is necessary to consider this disease separately as it involves the larynx and as it involves the naso-pharynx. For all therapeutical purposes we have practically two distinct diseases, although the cause may be the same. In the laryngeal type the danger is from asphyxia, either from laryngeal obstruction or, when this is overcome, from an extension of the membranous inflammation to the smaller bronchi; and the danger from sepsis is not great, because of the meagre lymphatic supply in this region and the small area of the surface from which absorption of toxins can take place.

On the other hand, in naso-pharyngeal diphtheria the danger from mechanical obstruction is slight, and the fatal cases are, almost without exception, the result of the absorption of poisons through the abundant lymph- and blood-supply. This is especially true of the nasal cases, as in this region not only is the blood- and lymph-supply very abundant, but it is almost impossible to obtain good drainage when the nasal mucous membrane and the turbinated bones are swollen.

Again, in laryngeal cases the disease is rarely the result of a mixed infection, but naso-pharyngeal diphtheria, as we see it in practice and not in the laboratory, is frequently due to a mixed infection. The importance of this from a therapeutical point of view is evident when we consider the difference between infection by Klebs-Löffler bacilli and by streptococci. The point is that in streptococcus infection the germ itself finds its way into the blood and viscera, but this is rarely true of the bacillus in Klebs-Löffler infection. In one case you have a toxin only to fight, and in the other you have both the germ and its toxin.

Although we admit that there are many unsolved therapeutical problems in connection with the antitoxin treatment of *naso-pharyngeal* diphtheria, there can be no doubt of its almost specific value in the *laryngeal* form of this disease. The laboratory proof is absolutely convincing as far as it goes—namely, that the serum in proper doses is a specific for preventing the harm which follows the absorption of the toxin of the Klebs-Löffler bacillus. The clinical results confirm this conclusion.

I can do nothing stronger to uphold this position than to give a short analysis of the cases of laryngeal diphtheria which I have seen during the past twelve years. I have arranged them from September to September, so that the cases of each winter will be kept together. With but few exceptions they have been seen in council with other physicians, and, since the antitoxin days, the diagnosis has been confirmed in nearly every case by a bacteriological examination by the New York or Brooklyn Board of Health.

Intubation Cases.

		No.	Recovered.	
July, 1885, to September, 1886,	1886,	37	7 = 18.9 per cent.	
Sept., 1886,	" 1887,	65	15 = 23.0	"
" 1887,	" 1888,	89	28 = 31.4	"
" 1888,	" 1889,	95	31 = 32.6	"
" 1889,	" 1890,	63	19 = 30.1	"
" 1890,	" 1891,	63	23 = 36.5	"
" 1891,	" 1892,	117	40 = 34.1	"
" 1892,	" 1893,	84	32 = 38.0	"
" 1893,	" 1894,	76	29 = 38.1	"
" 1894,	" 1895,	{ 13 with antitoxin . 5 = 38.4	"	Began calomel sublimations.
		{ 44 without " . 20 = 45.4	"	
" 1895,	" 1896,	{ 27 with antitoxin . 17 = 62.9	"	
		{ 3 without " . 0 = 0	"	
" 1896, to April, 1897,		{ 19 with antitoxin . 18 = 94.7	"	
		{ 1 without " . 0 = 0	"	
Total		796	284 = 35.6	"

The following table shows the results with and without calomel sublimations in all cases of laryngeal diphtheria up to September, 1894, or the beginning of the antitoxin treatment, and the results since the antitoxin was used:

442 cases; intubation; no calomel sublimations;	121 recovered = 27.3 per cent.
295 " " with "	123 " = 41.6 "
59 " " " antitoxin;	40 " = 67.8 "
50 " no " no calomel sublimations;	all recovered = 100 "
45 " " with "	" = 100 "
18 " " " antitoxin;	" = 100 "
38 died before my arrival.	
23 refused operation and died.	
21 died of sepsis with only slight obstruction.	
991 cases.	

It is interesting to note the steady improvement in results as our knowledge of the technique of intubation increased, and as we learned from experience to overcome, with greater success, the dangers and accidents of intubation. The marked improvement after calomel sublimations were used, and the still greater success after antitoxin, are noteworthy. This benefit is seen not only in the larger number of recoveries after operation, but in the increased percentage of cases which recovered without an operation. Thus of

492 cases, no sublimations, 50 recovered without operation = 10.1 per cent.
340 " with " 45 " " = 13.2 "
77 " " antitoxin, 18 " " = 23.3 "

Of course even this underestimated the good results, for the percentage of cases under calomel sublimations or the antitoxin treatment which recover without operation is very much larger. Since the introduction of antitoxin many cases recover and are never seen by the consultant which in former years would have undoubtedly come under his notice.

The apparently bad results after the use of antitoxin from September, 1894, to September, 1895, were probably due to two causes—inferior antitoxic serums and insufficient doses. A careful consideration of the cases during this period fails to show any marked difference in severity between those that received and those that did not receive antitoxin.

TREATMENT OF SEQUELÆ.—The treatment of the sequelæ and the albuminuria of diphtheria requires a few words. The albuminuria of this disease seems to be very little affected by treatment. The best that can be done is to

put the patient on a proper diet, compel the skin and the intestinal tract to do the work of the kidneys as much as possible, and to give a diuretic mixture—*e. g.* the infusion of digitalis with acetate of potassium. In bad septic cases the tincture of the chloride of iron seems to be useful; but marked diminution of urine, especially anuria, with a large amount of albumin, seems to be unaffected by any treatment, and usually ends fatally.

The anæmia should be treated by improving the nutrition and general condition in every possible way, and giving an iron mixture internally, a most satisfactory one being—

R. Tr. ferri chloridi fʒij to xij.
Glycerini fʒj.
Aquæ ad fʒiv.—M.

Sig.—Teaspoonful three times daily, in water, through a glass tube.

The chronic catarrh left after the disappearance of the pseudo-membrane should receive prompt and efficient treatment. In most cases the local application, continued for some time, of a weak solution of nitrate of silver will be all that is needed. But there are cases which may require operative interference and special treatment; and this treatment is discussed at another place in this work.

The natural tendency of the post-diphtheritic paralysis is to recovery. This is aided by every means which tends to increase the nutrition and improve the general condition. Therefore, good hygienic surroundings, plenty of easily-digested and nourishing food, iron, quinine, strychnine, and other tonics, are indicated. Strychnine, either hypodermatically or by mouth, seems to affect most beneficially the paralyzed muscles, one-sixtieth to one-thirtieth of a grain being given in twenty-four hours. Besides this, the careful use of massage and electricity does good service in assisting the nutrition and circulation of the affected muscles until the nerve-lesion gets well.

SYNOPSIS OF TREATMENT.—In brief, the treatment of diphtheria may be summarized as follows:

1. Put the patient in the best hygienic surroundings, with plenty of fresh air and sunlight. Keep the room at a uniform temperature of about 70° F., and give him an abundance of clean linen and bed-clothes. In protracted cases transfer the patient to a fresh room that has been thoroughly aired and not exposed to the disease, as many cases are undoubtedly liable to reinfection.

2. Keep up the strength and nutrition of the patient with plenty of stimulants and easily-digested and nourishing food.

3. Avoid all internal medication unless clearly indicated. The bichloride of mercury is useful, and in certain septic cases the tincture of the chloride of iron. The chlorate of potassium is dangerous.

4. Remove all broken-down membrane, pus, and other débris by irrigation of the diseased surface with a fifteen-volume solution of peroxide of hydrogen, diluted with lime-water.

5. To destroy the bacilli after the surface has been cleaned apply a solution of bichloride of mercury, 1:1000, either by irrigation or spray, and keep the room saturated with the vapor from a mixture of carbolic acid, turpentine, and eucalyptus. When it is impracticable to use the spray or irrigation, either from the location of the disease or the impossibility of managing the child, the best substitute is to make the patient inhale the fumes obtained by subliming calomel.

6. The treatment of the albuminuria is very unsatisfactory; in septic cases the tincture of the chloride of iron, in addition to the digitalis and acetate of potassium, gives the best results.

7. The sequelæ should be treated according to indications—the anæmia with iron and general tonics; the chronic catarrh by the application of weak solutions of nitrate of silver; and the paralysis by strychnine, massage, electricity, and general tonics.

8. Recent studies in immunity have given us a knowledge of an antitoxin which neutralizes or destroys the toxalbumin of the diphtheric bacillus. The following are the excellent rules for its use recommended by the American Pediatric Society at its meeting in 1897:

Antitoxin should be given at the earliest possible moment in all cases of suspected diphtheria.

Quality.—Of the products on the market some have, by test, been found to contain one-half to one-third the antitoxin units stated on the label. Select the most concentrated strength of an absolutely reliable preparation.

Dosage.—All cases of laryngeal diphtheria, the patient being two years of age or over, should receive as follows:

First dose—2000 units at the earliest possible moment.

Second dose—2000 units twelve to eighteen hours after the first dose if there is no improvement in symptoms.

Third dose—2000 units twenty-four hours after the second dose if there is still no improvement in symptoms.

Patients under two years of age should receive 1000 to 1500 units, the doses to be repeated as above.

TUBERCULOSIS.

BY WILLIAM OSLER, M. D.,
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I. GENERAL ETIOLOGY AND MORBID ANATOMY.

(a) INCIDENCE OF TUBERCULOSIS IN INFANCY AND CHILDHOOD.—Although it has long been known that, in the quaint language of Sir Thomas Browne, “consumptive and tabid roots sprout early,” the appreciation of the wide-spread prevalence of tuberculosis in the early periods of life is due to recent observations. Extremely rare in the new-born and uncommon in the first three months of life, the cases increase rapidly throughout the latter half of the first year and in the second year. In the *crèche* of the Hôpital Tenon of Paris, in the year 1890, it is stated that more than 21 per cent. of the babies died of tuberculosis. Of 2576 autopsies on infants made at Kiel, Boltz found 424 cases of tuberculosis. The following table gives the proportions at different ages:

Infants born dead	0.0 per 100	From 2 to 3 years	33.0 per 100
From 0 to 4 weeks	0.0 “ “	“ 3 “ 4 “	29.6 “ “
“ 5 “ 10 “	0.0 “ “	“ 4 “ 5 “	31.8 “ “
“ 3 “ 5 months	8.6 “ “	“ 5 “ 10 “	34.3 “ “
“ 6 “ 12 “	18.3 “ “	“ 10 “ 15 “	30.1 “ “
“ 1 “ 2 years	26.8 “ “		

The statistics of the late Professor Parrot embraced 219 cases in children under three years. Of these there were—

From 1 day to 3 months	23
“ 3 to 6 months	35
“ 6 “ 12 “	53

giving a total of 111 under one year of age, and from one to two years, 108.

Of 500 autopsies in children at the Munich Pathological Institute, Müller found tuberculosis in 150. Of 527 infants dead in hospital of various diseases, tubercles were present in 314.

A set of combined autopsies on 2230 children gave 753 tuberculous and 1407 non-tuberculous. The ages of the tuberculous cases are thus grouped:

From birth to 1 month	10
Up to 2½ years	138
From 3 to 5 years	255
“ 6 “ 10 “	226
“ 11 “ 15 “	124

Analogous statistics are not, to my knowledge, available in this country, but the observations of Northrup at the New York Foundling Asylum show, at any rate, that the disease must prevail quite as extensively. From the third to the fifteenth year tuberculosis is also very frequent, and its manifestations in the glands, skin, and bones contribute a very considerable percentage of all cases in the out-patient departments of hospitals and in the special infirmaries for children's diseases.

The mortality, highest in the first year, sinks rapidly throughout childhood, to rise after puberty. Thus of 10,000 living, there die (U. S. Census, 1870) of tuberculosis in the first year 18.5; in the second, 10.5; in the third, 5.9; from the third to the fifth, 2.9; from the fifth to the tenth, 2; from the tenth to the fifteenth, 3.3. The Kiel mortality statistics (Heller) also show this in a striking manner: of 10,000 living, there died in the first year 245; in the second, 114; in the third, 76; from the third to the fifth, 34; from the fifth to the tenth, 14; from the tenth to the fifteenth, 16.

(b) THE BACILLUS TUBERCULOSIS.—It is acknowledged by those most capable of expressing an opinion that the essential cause of tuberculosis is the organism discovered by Koch. The bacillus is a short, fine rod from 1 to 5 μ in length, and usually a little curved. In the sputum and in tuberculous tissue the bacilli are often in little clumps, or two lie crosswise at an acute angle.

For demonstrating the bacilli in sputa the following method will be found satisfactory: The thicker and more purulent parts of the sputum are picked out with a small sharp-pointed forceps and spread over the cover-glass, which is allowed to dry in the air and then passed three or four times through the flame. A few drops of Ziehl's solution of fuchsin—namely, distilled water 100 grams, carbolic-acid crystal 5 grams, alcohol 10 grams, fuchsin 1 gram—are placed upon the cover-glass, which is held over the flame until it begins to boil. The glass is then washed in water, and a few drops of Gabbet-Ernst's solution—namely, methylene blue 1 to 2 grams, 25 per cent. sulphuric acid 100 grams—are placed upon the glass and allowed to remain there for about a minute. The glass is then washed in water, and mounted either in water or, after drying between filter-paper, in oil or balsam. The tubercle bacilli are stained red, while the nuclei of the cells and any other bacteria are stained blue.

In sections the following method is pursued at the Pathological Laboratory of the Johns Hopkins Hospital: The tissues should be hardened in absolute alcohol and imbedded in celloidin. After the sections have been cut, the celloidin should be removed either with oil of cloves or with absolute alcohol and ether. After this they are passed through strong alcohol (to remove the oil or ether), and then placed in water previous to staining. The most satisfactory dye is the carbol-fuchsin solution of Ziehl. The sections are left for two hours at a temperature of 60° C. (or, if this be inconvenient, they may be stained for six or eight hours in the thermostat at 37° C., or for twenty-four hours at the room temperature). The tissue-elements and the bacilli are thus stained deeply in the fuchsin. A good decolorization solution is the ordinary acid alcohol of the laboratory (acid. hydrochloric. 1, aq. destill. 30, alcohol 70). The decolorizing process must be carefully watched, as too much of the dye may be easily extracted, the tubercle bacilli along with the tissue-elements losing their stain. It is best to remove the sections from the acid alcohol while they still retain a decided pink tint. A counter-stain is then used, the most desirable being a 2 per cent. aqueous solution of methylene blue. This removes all remaining fuchsin color from the tissue-elements and stains them a delicate blue. The tuber-

cle bacilli are stained a bright red. The sections are to be dehydrated in absolute alcohol, cleared in oil of cloves or preferably in xylol, and mounted in xylol balsam. It is best to examine with an oil-immersion lens, although if the bacilli are numerous they can readily be made out with a good high-power dry lens (Zeiss 3, or Leitz 7). Tubercle bacilli may be demonstrated in tissues by means of the rapid method used for staining them in sputum, but the results are very unsatisfactory, owing to the distortion of the tissues resulting from the action of the heat and the strong acids.

The bacillus is aerobic, and, although somewhat difficult to cultivate, may be grown on blood-serum, glycerin agar, or even on potato. The colonies form dry, grayish-white, scale-like masses. In the growth the bacillus forms certain soluble product or toxins, which, if introduced into the body, produce lesions similar to those induced by the bacilli themselves.

The bacilli are tolerably tenacious, and retain their virulence after freezing, desiccation, and salaison. It is stated that the bacilli have been found alive after burial of the subject for two years. The combined action of dryness and exposure to air is stated to diminish the virulence, but tuberculous sputum exposed to the air for from fifty to one hundred days still retains its virulence. The bacilli are rapidly killed in a few minutes by moist heat, as in boiling; dry heat is much less effectual. The bacilli are found in variable numbers in all tuberculous structures—the acute miliary nodule, the caseous, fibrous, and fibro-caseous nodules. They are most abundant in rapidly-growing tubercles and in the old ulcerous lesions of pulmonary tuberculosis. They are scanty, as a rule, in the more chronic tuberculous processes of glands and of bones, and in the lesions associated with extensive caseation. When not easily demonstrable by histological methods, inoculation in animals may alone determine the tuberculous nature of a structure.

Outside the body the bacillus has been shown to be a very widely-distributed organism, the number in any locality depending upon the number of cases of pulmonary tuberculosis and the carelessness or thoroughness with which the sputa of infected individuals is destroyed. In an ordinary case of pulmonary consumption countless millions are thrown out daily and scattered widely in the sputum dried as dust. Cornet found the dust of hospital wards and places occupied by tuberculous patients to be infective in a number of cases. Thus of 118 samples of dust from the wards of hospitals and rooms occupied by tuberculous individuals, 40 proved capable, when inoculated in animals, of producing tuberculosis. The infectiveness of the dust of the medical and surgical divisions of a hospital was found to be in the proportion of 76.6 to 12.5.

(c) MODES OF TRANSMISSION.—(1) *Experimental Tuberculosis*.—Much of our knowledge of the disease has been derived from experiments, and we owe to Villemin the demonstration of the infective character of all tuberculous processes. The receptivity of animals varies very much: the rabbit and guinea-pig are particularly susceptible; dogs and cats are very resistant. Bovines are very susceptible, and one of the most important facts in the etiology of the disease is the frequency with which the disease occurs in them.

Subcutaneous inoculation of tuberculous material in a susceptible animal, as a rabbit or a guinea-pig, is followed in a short time by the production of a little nodular growth, which softens, and even ulcerates, and which in time may be absorbed. The corresponding lymph-glands swell, tubercles develop in them, and then caseate. The animal dies in from six weeks to three months. Tubercles are found in the lymph-glands, and there is, as a rule, general tuberculosis of the organs. The most satisfactory method is the inoculation of

PLATE X.

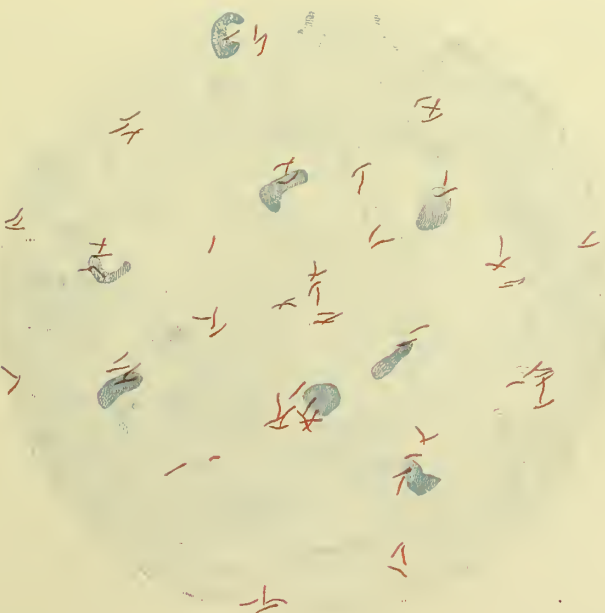
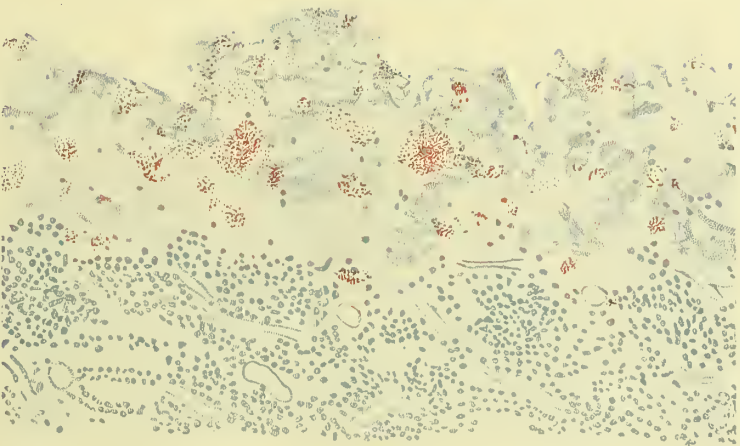
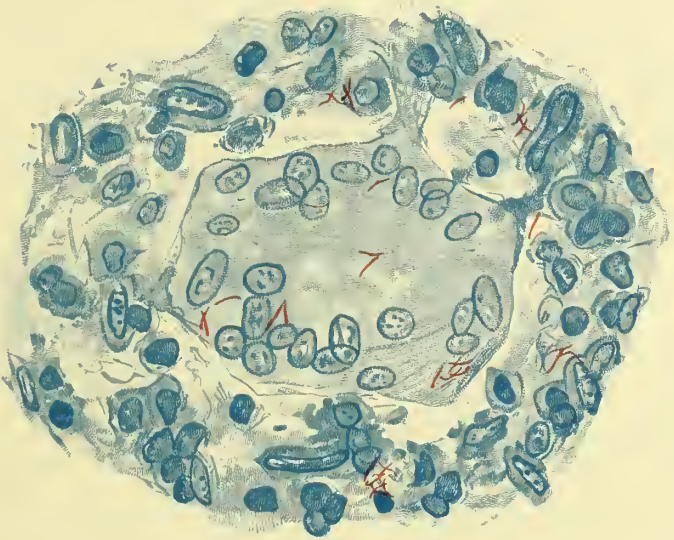


FIG. 1.—Section of a small fresh tubercle, showing large giant-cells surrounded by epithelioid cells. Tubercle bacilli are seen in the giant-cells and scattered in the tissue about it.

FIG. 2.—Section of tuberculous pleura under a low power, showing the exudate with groups of bacilli scattered in it.

FIG. 3.—Tubercle bacilli in sputum stained by the method given. The bacilli are more abundant than is usual.

material into the anterior chamber of the eye of the rabbit, as used by Cohnheim. The development of the tubercles, at first a local process, may be watched in the iris. There is afterward generalization, and the animal dies emaciated. In some instances in the rabbit and guinea-pig the lesion produced is entirely local and the animal recovers. If a culture of tubercle bacilli is injected into the veins, the animal dies, as a rule, in a shorter time, with the development of miliary granulations, particularly in the liver and in the spleen. If a larger quantity be injected, the animal may die of a profound infection before the tubercles become visible to the naked eye.

The transmission by inhalation is more difficult in animals, and the results of causing animals to breathe air charged with tubercle bacilli are discordant, but in some instances undoubted pulmonary infection and general tuberculosis have followed. Experimental infection through the digestive passages has also been demonstrated, particularly in the feeding of animals with the milk of tuberculous cows.

(2) *Hereditary Transmission*.—Current opinion on this point may be expressed as follows: While in a few rare cases tuberculosis is transmitted directly from parent to offspring, in the great majority of all cases the heredity does not relate to the transmission of the seed itself, but of a disposition of body, a type of tissue-soil favorable to the development of the disease in case of accidental infection.

Congenital tuberculosis has been observed in some six or eight cases. In the case of Charrin there was generalized tuberculosis in a foetus seven and a half months old, the mother of which died of phthisis. In Berti's case the child, born at term of a phthisical mother, died on the ninth day, and two small cavities were found at the posterior border of the lower lobe of the right lung, which were shown microscopically to be tuberculous. In Merkel's case the tuberculous mother died two days after confinement. The child had tuberculosis of the palate and an abscess of the left trochanter major. In Jacobi's case the foetus, born at the seventh month, had miliary granulations in the liver, peritoneum, spleen, and right pleura. In the case described by Sabouraud the child born of a tuberculous mother died on the eleventh day. The liver and spleen were tuberculous.

In all of the cases reported it was direct maternal heredity. The mode of transmission is not at all certain, but it is probably transmission through the placenta. Tuberculosis of the placenta is very rare. Lehman has recently reported an instance in a woman aged twenty-nine dead of acute tuberculosis in the eighth or ninth month of pregnancy. The foetus was not affected, but on both surfaces of the placenta there were a few grayish nodules, which showed the characteristic structure of tubercle, with bacilli. It has been shown also that the placenta of a tuberculous woman proved infective; and, indeed, it is stated that the amniotic fluid of a tuberculous subject may produce the disease in a guinea-pig.

There are several experiments (Landouzy and Martin, Birch-Hirschfeld, and Armanni), which indicate that the virus may be present in the foetus without the presence of actual tubercles, since they found that portions of the viscera of fetuses born of tuberculous mothers were infective to guinea-pigs.

A modified view of this direct heredity is advocated by Baumgarten, who holds that the virus is directly transmitted, but remains latent, and does not develop until some time after birth. In support of this he quotes the large number of cases of tuberculosis in the early months, the figures illustrating which have already been given. He also lays great stress upon the occurrence of tuberculosis in the bones and the joints of children, regions to which the

bacilli would not be likely to be conveyed in accidental infection. This post-natal development he regards as analogous to *syphilis hereditaria tarda*, and he suggests that the actively growing tissues of the child restrain or inhibit the development of the germs.

There is no evidence to show that a tuberculous father can directly transmit the disease. The experimental evidence is also negative. Gärtner (whose recent article on "Heredity in Tuberculosis" is the most important contribution made to the subject of late years) found that in rabbits and guinea-pigs, with artificially induced tuberculosis of the testes, and whose semen contained bacilli, the embryos were never infected. On the other hand, of 65 female guinea-pigs which had consorted with the tuberculous bucks, 5 presented genital tuberculosis, and of 59 female rabbits under similar conditions 11 became infected.

In support of the view that tuberculosis is hereditary great stress is laid naturally on the frequency with which a history of the disease is met with in the parents. The estimates of various authors on this point vary from 10 to 50 per cent. Of 427 cases at the Johns Hopkins Hospital, there were only 53 in which the mother was affected, 52 in which the father had had tuberculosis, and 105 in which sister or brother had had the disease. The fact that the children and relatives of tuberculous individuals are more directly exposed to contagion than other individuals renders it difficult, as Fagge remarks, to draw a clear line between heredity and accidental infection.

(3) *Inoculation*.—This is not very common in man, as the skin does not offer a very suitable soil for the development of the tubercle bacilli. This mode of infection is, however, seen in persons whose occupations bring them in contact with dead bodies and animal products. Demonstrators of anatomy are particularly subject to a local tubercle on the finger or back of the hand—the so-called post-mortem wart, *verruca necrogenica*, the "lichen" tubercle of the Germans. Only in very exceptional instances is this followed by serious results. Cases have been reported of infection from the bite of a tuberculous patient, inoculation from a cut by a broken spit-cup and the puncture of a hypodermic needle. There is no reliable observation of the transmission of tuberculosis by vaccination. In the performance of the rite of circumcision children have been inoculated, the infection in these cases being associated with disease in the operator, and occurs in connection with the habit of cleansing the wound by suction.

(4) *Transmission by Inhalation*.—The expired air of tuberculous patients is harmless, but the sputa, dried and widely diffused in the form of dust, constitute one important medium of transmission in the disease. The investigations of Cornet have shown the greater infectiveness of the dust of localities frequented by patients with pulmonary tuberculosis. The frequency with which the disease is met with in the lungs and in the bronchial glands finds here its explanation.

In institutions the residents of which are restricted in the matter of fresh air and exercise, as in jails and convents, the death-rate from tuberculosis is very much higher than in the general population. Cornet found that in some of the religious communities more than three-fourths of the deaths were due to this disease. The mortality in prisons from tuberculosis is from 40 to 50 per cent., while in the general community it is not more than 15 per cent. Flick has brought forward evidence to show that the distribution of tuberculosis in one of the wards of the city of Philadelphia is more particularly with certain houses in which individuals have died of this disease. There are also some striking local epidemics of tuberculosis: thus Marfan gives an instance of a

place confined and badly ventilated, occupied by twenty-two employees, who were joined in 1878 by two consumptives, who for several years coughed and spat about the floor indiscriminately. The employees arrived at an early hour and breathed the air charged with the dust raised by the morning cleaning. Between 1884 and 1889 thirteen of these persons fell victims to tuberculosis.

Against these facts, however, are the statements that at hospitals for consumptives, as at Brompton, in London, the doctors and nurses are rarely attacked. Dettweiler claims that at his institution in Falkenstein no case of tuberculosis has been contracted. On the other hand, Marfan states that in the Paris hospitals tuberculosis is extremely frequent in the attendants and decimates the lay contingent. At the Hospital Necker half of the attendants are attacked with phthisis, and he notes as a significant fact that it is particularly the attendants in the medical wards.

The danger is enhanced when the contact is particularly intimate, as between a tuberculous mother and her child or between man and wife. In the latter case there are figures which indicate that contagion is not at all infrequent.

(5) *Transmission by the Food.*—Experiments have shown that infection may be communicated by ingestion of tuberculous material, and one of the most important problems relates to infection with the milk of tuberculous cows. Experimentally, it has been conclusively demonstrated that such milk is infective, even when the disease is localized in the lungs of the animal, and that it is not necessary that the udder should be diseased. Ernst has shown that the bacilli may be present in the milk when there is no tuberculous mammitis. The danger of infection from this source in children is very urgent, and systematic sanitary inspection should be made of the cows, and, if necessary, inoculation experiments made with the milk.

The percentage of tuberculous animals in the dairy-stables of our cities is very much larger than has been supposed. The figures in this country for large numbers are not available. It has been stated that from 10 to 15 per cent. of the dairy stock in the Eastern States is tuberculous. This is probably a low estimate.

The virulence is retained in the cream and in the butter. Other conditions than the presence of the bacilli in the milk are probably necessary for infection, and, fortunately, all children who drink tuberculous milk do not become contaminated. In some instances the gastric juice may destroy the bacilli; in others, conditions of the tissues may not be favorable to the development of the seed. Experimentally it has been shown that lesion of the intestines itself is not necessary, and infection of the mesenteric glands may take place through a normal mucosa. Possibly the great frequency of mesenteric tuberculosis in children finds here its explanation. In 127 cases of fatal tuberculosis in children noted by Woodhead these glands were involved in 100. It is not definitely determined whether the milk of a tuberculous woman is virulent.

Infection by meat is probably very much more rare. When the tuberculosis is generalized in the internal organs the flesh should be confiscated. The virulence, however, is only marked when the disease is very extensive. It has been shown that the flesh of tuberculous subjects is infective to guinea-pigs. Nocard, however, in a series of experiments found that the juices of the muscle of twenty-one cases with general tuberculosis, when injected into the peritoneum of guinea-pigs, only once produced tuberculosis.

(d) CONDITIONS INFLUENCING INFECTION.—(1) *General.*—These, dealing specially with the environment of individuals, explain in a great measure the

frequency of the disease in certain localities. Thus one of the most important is residence in the large centres in which many people are crowded together. The death-rate from tuberculosis is very much higher in towns than in the country, and a very considerable share of the high infant mortality of cities is to be attributed to it. Not only is the air of the large towns less pure, but the chances are very much greater that the dust, blown in all directions, has with it the germs of the disease. The inhalation of impure air in certain occupations, which in adults is an important predisposing factor in pulmonary tuberculosis, does not prevail to any special extent in children. Climate in itself does not influence the conditions materially, but, as a rule, the disease is more common in the temperate regions, largely because in these are found the largest collection of human beings. Soil and locality have an important influence, cold and dampness increasing the personal liability by favoring the development of catarrhal affections. There are fewer cases of tuberculosis and fewer foci of infection in regions such as the Alps and in elevated plateaux as in Mexico, but altitude itself does not confer immunity, and there are many mountainous regions in which the inhabitants are much affected by tuberculosis.

More important than these are the factors relating to personal environment, as of the dwellings. The constant breathing of a vitiated air, as in the small crowded rooms in the tenements and narrow alleys of our large cities, and the absence of sunlight, are two of the most important predisposing elements in tuberculosis in children. These influence infection in two ways: first, by favoring the distribution of the bacilli; and, second, by lowering the nutrition of the individual and leading to conditions favoring the entrance of the bacilli to, or their development in, the body.

(2) *Individual Predisposition*.—From the time of Hippocrates it has been thought that there was a certain conformation of body which rendered an individual more prone to the disease. His words are: "The form of body peculiar to subjects of phthisical complaints was the smooth, the whitish, that resembling the lentil; the reddish, the blue-eyed, the leuco-phlegmatic; and that with the scapulæ having the appearance of wings." In children it may be said that the build and type such as here described is certainly more prone to tuberculous affections. Two types of conformation have long been recognized as predisposing in some way to infection; the *tuberculous*, with bright eyes, oval face, thin skin, and long thin bones, and the *scrofulous*, with a heavy figure, thick lips and hands, opaque skin, and large thick bones. But, as in adults, well-developed, healthy infants and children may become subject to the disease. In addition to the conformation of the chest, the respiratory capacity, the relation between the volume of the lung and of the heart, a relatively small heart with narrow arteries, and a pulmonary artery relatively wider than the aorta (Beneke), and relatively large-sized viscera, have all been brought forward as causes predisposing to tuberculosis.

Among others which may be mentioned is race: the negro seems more liable to the disease than the white races, and it is stated that the Hebrews possess a relative immunity.

More important in children are the local conditions influencing infection. Acute and chronic catarrhal troubles of the throat and upper air-passages, and of the lung, undoubtedly favor infection, either by allowing the freer entrance of the germs or by weakening the powers of resistance. The infectious diseases, particularly whooping-cough, measles, and influenza, act probably in this way, while small-pox, typhoid fever, and syphilis influence the conditions rather by reducing the power of resistance. In institutions the frequency with which tuberculosis follows the infectious disorders is very striking.

Of local affections of the lungs which predispose to tuberculosis, hæmoptysis, which was regarded as an important cause, is now thought to be an indication rather of the existence of the disease. Such disorders as dilatation of the bronchi and pleurisy also heighten the liability to infection, though in the latter case many of the instances believed to be simple are in reality from the outset tuberculous.

The subjects of congenital or acquired contraction of the orifice of the pulmonary artery usually, as is well known, die of tuberculosis. Prior to the development of the disease many subjects show a marked anæmia, and unquestionably chlorosis offers favoring conditions for the development of this affection. Diseases of the stomach and intestines, particularly chronic entero-colitis, increase the susceptibility to infection.

Blows and contusions favor in some way the development of tuberculosis, particularly in children, in whom spinal caries and hip disease may follow an injury; less often does trauma play any part in pulmonary tuberculosis. Here, too, may be mentioned the favoring influence of operation: resection of a tuberculous joint is occasionally followed by an acute infection.

With reference to infection and the conditions which influence it the following may be stated:

(a) In a few cases the disease is directly transmitted from the mother, and appears in the child at birth.

(b) The primary tuberculosis of the bones, joints, kidney, spleen, liver, etc. of early youth is very possibly associated with a foetal hæmatogenous infection (Baumgarten, Gärtner).

(c) Direct paternal transmission has not been proved, and experimental evidence is strongly against it.

(d) In a large proportion of cases the infection is post-foetal—through the lungs, intestines, or skin.

(e) Heredity influences the *soil*. All are *tuberculizable*, to use a French expression, and very many of us actually become infected. Whether or not the *seed* develops depends, firstly, upon the character of the tissue-soil; and secondly, upon the existence of special favoring circumstances.

(f) Immunity, a relative condition, enjoyed chiefly in consequence of inherited tissue-resistance, is lessened by all circumstances which depress nutrition, such as bad air, bad food, and imperfect hygienic surroundings. Next to the germ, a vulnerability of tissue, however brought about, whether congenital or acquired, is the most important factor in the etiology of the disease.

(3) *The Relations of Tuberculosis and Scrofula*.—The lesions known as scrofulous are tuberculous, and due to the development of the *bacillus tuberculosis*, so that the term scrofula is now almost, and may well be entirely, abandoned. Though the so-called scrofulous lesions of glands and bones and skin are bacillary in origin, yet it has been shown that their virulence is not so extreme as that of the tuberculous products in the viscera, the latter, according to Arloing, killing, when injected, both guinea-pigs and rabbits, the former only guinea-pigs. This corresponds with the more protracted course and the more favorable termination of the so-called scrofulous affections. It has been suggested that the scrofulo-tuberculous manifestations are caused by an attenuated virus. An attempt has been made by writers, particularly Marfan, to show that the scrofulo-tuberculous lesions, when recovered from, confer a sort of immunity to pulmonary tuberculosis, but the evidence for this is not yet very strong.

(e) ANATOMICAL CHANGES PRODUCED BY THE TUBERCLE BACILLI.—The lesions induced by the bacilli are in the form of small nodules which, fused

together, may form large infiltrated areas, so that a distinction is often made between the nodular and the diffusely infiltrated varieties. The studies of Baumgarten and others have enabled us to follow accurately the primary changes induced by the bacillus. These are—

(1) The multiplication of the fixed tissue-elements by a process of karyokinesis. The cells of the vascular epithelium, of the ordinary epithelium, and of the connective tissue proliferate, and gradually there is produced from them the large, rounded, cuboidal, or polygonal cells with vesicular nuclei—the so-called *epithelioid* cells—inside some of which the bacilli are seen. This reaction of the fixed elements of the tissue would appear to be the primary effect.

(2) Leucocytes, chiefly polynuclear, migrate, and accumulate about the focus of infection. These form the lymphoid cells which were formerly thought to be so characteristic of the tubercle. They do not, however, undergo subdivision. Some believe that they attack and destroy the bacilli. There would appear to be successive invasions of leucocytes at the focus of irritation, and many of them undergo rapid destruction. It is stated, too, that later, as the little tubercle grows, the leucocytes which surround it are of the mononuclear form, or lymphocytes, and that these persist and do not undergo the rapid degeneration of the polynuclear forms.

(3) A reticulum of fibres is formed in the tubercle by the fibrillation and rarefaction of the connective-tissue matrix, most apparent, as a rule, at the margins of the growth.

(4) In some, but not in all, tubercles giant-cells are formed by an increase in the protoplasm and in the nuclei of an individual cell, or possibly by the fusion of several cells. The bacilli are usually, but not always, seen in the giant-cells. There seems indeed to be an antagonism between the number and virulence of the bacilli and the giant-cells: thus in joint and gland tuberculosis and in lupus, in which the former are scanty, the latter are abundant; while in miliary tubercles, and, as a rule, in all lesions in which the bacilli are abundant, the giant-cells are scanty.

The tuberculous nodule thus formed may undergo necrosis and caseation, or may gradually be converted into a connective-tissue mass. Caseation begins at the central part of the growth, and appears to be owing to the direct action of the bacilli. The cells undergo coagulation necrosis, lose their outline, become irregular, and are finally converted into a homogeneous, structureless material in which the cells are no longer distinguishable, and which no longer takes the stain. As this process extends involving several nodular tubercles, they are gradually converted into uniform yellowish-gray masses. No blood-vessels are found in the central portion, but the bacilli are usually abundant. By the union of many of the nodular tubercles large masses may be formed which may undergo either (1) softening or liquefaction with the formation of cavities; (2) fibroid limitation, leading ultimately to encapsulation; (3) in the older caseous masses, particularly when encapsulated, lime salts may be deposited (calcification); and (4) sclerosis. There is necrosis of the tissue-elements in the centre, gradual hyaline transformation, with great increase in the fibrous reticulum, so that the tubercle is ultimately converted into a firm, hard structure. Sometimes increase in the fibrillation and caseation go on together, with the production of fibro-caseous tubercle.

Diffuse Infiltrated Tubercle.—It was formerly thought that the products of any simple inflammation might become caseous, and the identity of the caseous pneumonia and of scrofulous lesions with tubercle, which Morton (1685) maintained, and which Laennec laid down as a fundamental proposition, was for a long time disputed, particularly by Virchow. Now, the researches of Koch

have demonstrated that these infiltrated caseous lesions are definitely tuberculous.

Infiltrated tubercle results from the fusion of many small nodular foci, too small sometimes to be visible to the naked eye. Histologically, however—in the lungs, for instance—they may be seen to be composed of scattered centres surrounded by zones in which the air-cells are filled with leucocytes and the products of the proliferation of the alveolar epithelium. Under the influence of the bacilli caseation takes place, usually in small groups of lobules, but occasionally in an entire lobe, or it may be throughout the greater part of a lung. There is really no essential difference between the nodular and the infiltrated tubercle.

Secondary inflammatory processes accompany the growth and development of tubercle: (1) The exudation of leucocytes and serum about the primary growth is in reality an inflammation, which varies with varying conditions, and which may be limited or very extensive. For example, about the tubercles in the lungs there is always inflammation of the alveoli with infiltration and proliferation of the connective-tissue elements of the septa, and changes in the blood- and lymph-vessels.

(2) The bacilli themselves may induce suppuration, as in joint and bone tuberculosis; experimentally, the products of the growth of the tubercle bacilli, particularly Koch's tuberculin, produce an active suppuration. In tuberculosis of the lungs, as well as in other regions, the suppuration is largely the result of a mixed infection, and is due to pus-organisms.

(3) A slow, reactive inflammation occurs about many tubercles, resulting in the formation of a cicatricial connective tissue, limiting and restricting their growth, and constituting, in reality, the important conservative and healing element in the disease.

II. GENERALIZED FORMS OF TUBERCULOSIS.

(1) ACUTE MILIARY TUBERCULOSIS.

FORMS of tuberculous infection running a rapid course are decidedly more common in infants and children than in adults. Practically, there is always a focus of local disease in a bronchial or mesenteric gland, a joint, or on the skin, or in superficial lymph-glands. In a few rare instances a miliary tuberculosis is encountered in which caseous foci cannot be discovered. The picture may be either that of an acute infection without definite local manifestations, or of an intense infection with pronounced symptoms pointing to involvement of the meninges of the brain, the lungs, or the serous membranes. In children there is no hard-and-fast line to be drawn between the acute forms in which miliary granulations occur throughout all the organs, and in which the clinical course is from three to six or eight weeks, and forms in which throughout the various organs there are coarser, larger grayish-yellow tubercles, and in which the clinical course is of more subacute character, lasting from eight to twelve or even sixteen weeks. As in the adult, the cases may be divided for convenience into three groups, as the symptoms are those of a general infection, simulating very often typhoid fever, or those of an acute meningitis or of an acute affection of the lungs. These cerebral, general, and pulmonary types cover a majority of the cases. There may be mentioned, in addition, an acute affection, occurring in children the subjects of a local tuberculous process, in which, with the symptoms of a profound infection, there is no general miliary tuberculosis. This form, which has been described by several French writers as the *fièvre*

infectieuse tuberculeuse suraiguë, is not generally recognized, but the cases described by Landouzy and others presented quite minor tuberculous lesions of the lungs or of other organs, with the clinical symptoms of very intense infection, the severity of which was out of all proportion to the local lesion and to the number of miliary granulations found throughout the body. It is thought to be due to the action of the tuberculous toxines.

The acute tuberculous meningitis will be described separately. We shall here speak only of the typhoid and of the pulmonary types.

Typhoid Type.—The onset is usually insidious, and commonly there has been an indisposition or slight cough, but prior to the fever the child may have been in good health. The fever is noticed in the afternoon or evening, and with it there is loss of appetite, and the child loses in weight and is listless and not inclined to play. A bronchial cough is by no means uncommon, but it is to be remembered that the disease may set in quite abruptly in a child believed to be in good health. Within a week, or even earlier, the child takes to bed, and the symptoms of an infection are well pronounced. The tongue is white and furred. The abdomen is distended, sometimes painful on pressure, and there may be diarrhoea. The spleen is usually enlarged, and can be readily felt. The liver may be also distinctly swollen. The gastro-intestinal trouble with the continued fever may be strongly suggestive of typhoid fever, but rose-spots are not detected. There are usually some symptoms pointing to the involvement of the bronchi or the lungs, and the respirations are hurried, only, however, in proportion to the fever, and the physical signs indicate rather a catarrh of the larger than of the smaller tubes; there is no dulness. There are headache, delirium, particularly at night, and sometimes marked hyperæsthesia of the skin. Albuminuria is often present, and there may be complete suppression of urine. The fever varies greatly in intensity, but usually has not the regularity of typhoid, and the daily exacerbations are more marked. It may rise to 104° and 105° F. On the other hand, there are cases in which the fever is moderate, not more than 101° or 102° , and very rarely there may be no fever. There are also instances in which there have been rigors throughout the course of the disease. The condition of the child becomes aggravated, and with a dry tongue, delirium, unconsciousness, distended abdomen, and swollen spleen, the similarity to typhoid fever is very close. The course is extremely variable, and while death may occur at the end of the second or beginning of the third week, in other cases the disease is prolonged to five or six weeks. In the more protracted cases definite local signs are met with; thus, with an increase in the dyspnoea and cough bronchitis of the smaller tubes is found, and patches of consolidation at the bases, so that aëration is very defective. The eruption of tubercles on the meninges may intensify the cerebral manifestations, and there may be from the outset severe headache, with a gradual and progressive coma, dilated pupils, and sometimes strabismus.

Pulmonary Type.—The clinical features are of an intense capillary bronchitis (broncho-pneumonia). This, the more common variety, is very often mistaken at its onset, and even throughout the course, for simple broncho-pneumonia. The onset may be abrupt, and even with a chill, but, as a rule, the child has been failing in health or is at the time convalescing from some acute illness or is the subject of an acute naso-pharyngeal catarrh. The fever is high, and may reach from 103° to 105° ; the pulse is rapid, from 130 to 140. The respiratory symptoms are marked. At first the shortness of breath is slight and proportionate to the fever, but gradually it increases, and the respirations may be from 60 to 70 per minute. The cough is frequent, dry, and very troublesome. As the dyspnoea becomes more marked the color of the face changes,

and there is slight cyanosis. Though the fever is high and the symptoms grave, there are rarely severe cerebral manifestations. There may be slight diarrhoea, but the abdomen is not specially distended; the spleen is easily palpable. The whole clinical picture is that of an acute broncho-pneumonia. The physical examination shows hurried respiration, and there may be retraction of the lower zone of the thorax; the percussion note is clear, even hyperresonant, and auscultation at first shows signs of a general bronchial catarrh, chiefly of the smaller tubes. Subsequently, as the case progresses, there are areas in which the resonance is higher in pitch or even tympanitic, and in places distinct blowing breathing may be heard, or even the signs suggestive of cavity.

The course of the disease in this type is much more rapid, and the child may die at the end of a week, or even earlier, with the signs of an acute suffocative catarrh—more commonly in from ten to twelve or fourteen days, usually from a progressively advancing asphyxia.

Diagnosis.—The diagnosis of acute tuberculosis in children may be very easy or beset with the greatest difficulties. The family history should be taken into account; the surroundings of the case, particularly whether there have been instances of tuberculosis in the same house or occupying the same room. Much more important is the previous history and personal condition of the patient. Inquiries should be made about whooping-cough and measles, diseases not infrequently followed by acute tuberculosis. Sometimes a history of failing health or of protracted catarrh may be obtained. The most evident cases are those in which there are signs of local glandular or bone tuberculosis. Sometimes the acute affection follows an operation on the tuberculous glands of the neck or the opening of a joint abscess, or even of a so-called cold abscess, or, in very rare instances, the tapping of a pleural effusion. In the typhoid type, when the features are well developed, the simulation of ordinary enteric fever may be extremely close. Here, if from the outset a careful temperature record be kept, it will usually be found that the fever is much more irregular in tuberculosis, and early in the disease there may be quite marked morning remissions. As noted before, in a few instances the temperature may be low, even subnormal, in the morning. The general features of infection are much the same in both diseases. The absence of typhoid rash, unless it is there, which is usually present in children, and very distinctive, is a most important negative sign. Expectoration is rarely obtained, but should the child vomit, sputa should be looked for in the vomitus, since it sometimes happens that an acute miliary tuberculosis takes its origin in a small focus of disease in one lung, from which tubercle bacilli may reach the sputum.

The examination of the urine is important, but Ehrlich's reaction is present as frequently in acute tuberculosis as in typhoid fever. Pus in the urine should be carefully examined for bacilli, since instances of general infection have resulted from urogenital tuberculosis.

The profound infection associated with malignant endocarditis may simulate that of acute tuberculosis. The special heart-signs, if present, and embolic features, would be important distinguishing marks. The diagnosis of the catarrhal or broncho-pneumonic type will be more fully considered when speaking of the acute tuberculous broncho-pneumonia of infants.

Prognosis.—The prognosis is always unfavorable. Here, however, may be mentioned a type of acute tuberculosis recognized by Empis, Landouzy, and others, which they call *typho-tuberculose* or *typho-bacillose*, and which may be either the first manifestation of the invasion of the organism with the bacilli or the expression of an acute, but aborted, tuberculosis, following some local tuberculous process. The clinical aspect is really that of typhoid fever, and

the temperature curve would not appear to give any definite criterion. Unless, in fact, there is some local tuberculous focus, I do not see how this form can be recognized, and many of the cases reported by Aviragnet in his monograph are not at all convincing. That there may be, however, either early in a tuberculosis, or as a secondary event in a local process, an infection of the system with the toxins is extremely likely. In adults it is not very uncommon to find a tuberculous focus completely overlooked in a general infection believed to be typhoid fever, and in which the secondary development of miliary granulations seems scarcely sufficient to account for all the symptoms.

(2) CHRONIC DIFFUSE TUBERCULOSIS.

This, one of the most common forms of tuberculosis in children, is characterized anatomically by the gradual development of tubercles in many different parts of the body: they are not, however, the miliary granulations of the acute tuberculosis, but coarse, grayish-yellow tuberculous masses, varying in size from a pea to a walnut. In the lungs, for instance, there are caseous tubercles of all sizes, areas of caseous broncho-pneumonia, some of which have undergone softening; but cavities are not common except in children above four or five. The bronchial glands are often greatly enlarged and caseous, and sometimes present abscesses. The abdominal organs show extensive tuberculosis. The spleen is greatly enlarged, and on section presents numerous grayish-yellow tuberculous masses, varying in size from 2 to 10 mm. The liver is enlarged and may show miliary tubercles on the capsule, but in many instances there are coarser yellowish-gray masses which have developed about the bile-capillaries, and which, having softened in the centre, present a yellowish-green bile-stained pus. The small intestines may show tuberculous ulceration to a greater or less extent. The mesenteric glands are usually enlarged and caseous. The kidneys may show coarse tubercles, sometimes an intense tuberculous pyelitis. In the brain there may be either an acute terminal meningitis or there are coarse tuberculous nodules scattered throughout the substance, particularly in the cerebellum. The chronic diffuse tuberculosis is much more frequent in infants than in children above the age of two. The symptoms are those of a progressive enfeeblement of the nutrition, as a rule *without fever*, and with manifestations in different organs varying with the degree of tuberculization. The affection may set in acutely as a bronchitis or a broncho-pneumonia, the symptoms of which gradually subside. Very often the condition follows whooping-cough, measles, or acute gastro-intestinal catarrh. Less frequently it is insidious, and the child presents simply progressive failure in health. The appearance of the child is that of marked cachexia. It is thin; the skin is loose and pale, sometimes covered with fine scales, and occasionally pigmented. The eyes are large, and the expression often bright and animated. The thorax is thin, the ribs readily noted, and there may or may not be the signs of coexisting rickets. The abdomen is usually tumefied, and both the liver and spleen are enlarged. When the abdominal features are marked, the clinical picture is that really of some cases formerly described as *tabes mesenterica*. The superficial glands may be enlarged and hard. Cough may be present, usually dry, and very rarely there is dyspnoea. The physical signs throughout the lungs are either dulness in the interscapular regions or scattered areas of defective resonance with bronchial râles and blowing breathing. The appetite is poor, the digestion feeble, vomiting is frequent, and diarrhoea is common. Not only may there be no fever, but the temperature may even be subnormal. Death usually results from some complication, either a secondary invasion of pneumococci or streptococci, or an acute meningitis.

The diagnosis may present difficulties if one does not constantly bear in mind, in the first place, the frequency of tuberculosis in infants, particularly in institutions; and, secondly, the fact that this diffuse form, which is very common, may pursue its course without fever, and only perhaps toward the close show signs of active disease, now of the meninges, now of the lungs, or, again, of the intestines. This cachexia of the chronic diffuse tuberculosis of infants must be distinguished from that of rickets, of chronic gastro-intestinal catarrh, and of syphilis. In rickets the changes in the bones and cartilages, in athrepsia the marked gastro-intestinal disturbance, and the, as a rule, more enfeebled and senile look of the child, serve as distinguishing features. The absence of enlargement of the spleen and liver or of the lymph-glands is an important negative sign. A greater difficulty exists in distinguishing some of the cases of profound syphilitic cachexia, as here the superficial glands may be enlarged and the spleen and liver hypertrophied; but, on the other hand, the history, the facies, the skin-rashes, rhagades, and, above all, the prompt improvement under antisyphilitic treatment, are important points of differentiation.

III. LOCALIZED TUBERCULOSIS.

(1) TUBERCULOSIS OF THE LYMPH-GLANDS.

(a) TUBERCULOUS POLYADENITIS.—The lymphatic system may be the chief seat of the disease, and the glands, internal and external, or the lymph-sacs (serous surfaces), may present advanced tuberculosis without much involvement of the viscera or other parts. This is more often the case than we have heretofore supposed. In some instances of general tuberculous infection in young children there may be what Legroux calls micro-polyadenopathy, which in doubtful cases may give an important diagnostic hint. More recently Lesage and Pascal have described cases in children in which there was progressive involvement of the lymphatic glands, usually at first those of the groin, then those of the axilla, and lastly the cervical and internal groups. They regard the affection in some of the cases as due to cutaneous tuberculosis; in others they believe the disease to be congenital. The symptoms of this form of generalized enlargement of the superficial lymph-glands are progressive cachexia without much fever and without signs of disease of the lungs or of the abdominal organs, and frequently a ravenous appetite.

The cases must be carefully distinguished from the general slight enlargement of the lymph-glands in syphilis, and from the rare cases of Hodgkin's disease in children, in which, however, the enlargement is much greater and the involvement of one group is generally much more marked. It must not be supposed, however, that every case of general moderate enlargement of the superficial lymph-glands in children is due either to tuberculosis, syphilis, or Hodgkin's disease. Following the infectious fevers, and associated with chronic catarrh of the upper air-passages, I have seen on more than one occasion enlargement of the glands of the neck, of the groin, and of the axillæ—a condition of the superficial lymph-apparatus comparable to the swelling of the Peyer's follicles and of the mesenteric glands found so frequently in children dead of one of the infectious diseases, or, in fact, of any prolonged illness.

(b) CERVICAL ADENITIS.—The drainage-areas of the lymphatic glands of the neck embrace the superficial and deep structures of the head and neck.

The most important groups are the superficial cervical, beneath the platysma, which drains the side of the head and neck and face and external ear, and the deep cervical along the carotid sheath, which drains the mouth, the tonsils, palate, pharynx, and larynx. In addition there are the submaxillary and suprahyoid groups draining the lower gums, the front of the mouth and tongue, and the chin and lower lips.

Tuberculous adenitis of the glands of the neck, so extremely common, which fortunately often remains a local and curable affection, was regarded as one of the most typical and characteristic manifestations of scrofula. Cornet's observations upon the presence of tubercle bacilli in the dust of cities and of rooms show how widely spread the virus is, and how liable we are in crowded cities to inhale, and even to swallow, bacilli with the dust. Whether the bacilli are capable of passing through the healthy mucous membrane is perhaps doubtful, though there are experiments which would seem to prove the liability of infection through the healthy mucous membrane of the intestines. More probably the slight catarrhal troubles about the naso-pharynx, so frequent in children, open, as one may say, the portals and allow the bacilli to reach the lymph-glands. Preliminary irritation and enlargement of the glands in eczema of the scalp and in sore throat in children may weaken the powers of resistance. Here, no doubt, if the tissue-soil be unfavorable, they may exert no influence whatever, but with that vulnerability of tissue, regarded by former writers as the characteristic feature of scrofula, the bacilli find a suitable nidus, and a local tuberculosis is the result—a process characterized usually by extreme chronicity.

The glands may enlarge rapidly at first and become soft and painful; more commonly, they swell slowly, and can be felt as firm rounded masses freely movable beneath the skin. They may gradually subside and undergo spontaneous healing. In other instances the glands increase, areas of softening are found, the process involves the skin overlying the gland, which becomes red, and finally ulcerates, discharging a cheesy matter and a thin watery sero-pus. The sore thus left is very indolent, does not tend to heal; the skin about it is livid and undermined. Many of the glands may suppurate in this way, and when healing ultimately takes place the sides of the neck are disfigured by irregular, unsightly scars. In the neck of young or old these are usually a certain sign of healed tuberculosis.

It is to be borne in mind that involvement of the cervical glands may be due to extension of tuberculous processes from the axillary glands or even from carious cervical vertebræ. When the glands are large and growing actively there is fever; death very rarely follows, and even aggravated cases in children may recover. In some instances the general nutrition is very slightly disturbed. Tuberculous adenitis of the cervical or axillary groups may precede the development of tuberculosis of the pleura or of the lung.

(c) *TRACHEO-BRONCHIAL GLANDS.*—Within the thorax the groups of lymph-glands are of great importance. The sternal are placed along the course of the internal mammary vessels; the intercostal along the heads of the ribs, and sometimes extending outward; the anterior mediastinal group between the lower part of the sternum and the pericardium; the cardiac group in the interpleural space about the arch of the aorta; and, lastly, the tracheal glands on either side of the windpipe, and the bronchial proper, continuous with them, which surround the main bronchi and pass deeply in the hilus of the lung. There are also glands in the posterior mediastinum along the thoracic aorta and œsophagus. Tuberculosis of the tracheo-bronchial glands is extremely

common. Observations of Loomis (Jr.) show even that in apparently normal glands bacilli may be present and the gland-tissue infective. Certainly in a very large proportion of all cases of tuberculosis in children it would appear that the first infection was in these structures, while common experience shows, contrary to the so-called law formulated by Parrot, that the glands may be involved without any local lesion in the lungs. Of 125 cases examined by Northrup, the bronchial glands were tuberculous in every case; 42 had cheesy masses in the bronchial lymph-nodes only, with recent tubercles in the lungs and elsewhere; in 13, it was limited to the bronchial glands alone. The glands may present gray miliary tubercles, large, unpigmented, cheesy areas, foci of softening with suppuration, or old calcified masses. In the long-standing cases there is much sclerosis and pigmentation. The different groups may be very differently involved; thus the tracheal may be much affected without great involvement of the bronchial nodes proper. More commonly all the glands are involved, and very often those deep in the hilus of the lung form large caseous masses uniformly surrounding the main bronchus and its divisions, and penetrating deeply between the lobes of the lung. When the glands suppurate the abscesses may perforate in different directions. The effects of these enlarged glands are very varied, and for full details the reader is referred to the elaborate section in the *Traité de Barthez and Sanné* (tome 3). It is sufficient here to say that there are instances on record of compression of the superior cava, of the pulmonary artery, and of the azygos vein. The trachea and bronchi, though often flattened, are rarely seriously compressed. The pneumogastric nerve may be involved, particularly the recurrent laryngeal branch. More important, really, are the perforations of the enlarged and softened glands into the bronchi or trachea, or a sort of secondary cyst may be formed between the lung and the softened bronchial gland. Perforations of the vessels are much less common, but the pulmonary artery has been opened. Perforation of the œsophagus has been described in several cases. One of the most serious effects is infection of the lung or pleura by the caseous glands situated deep along the bronchi. The infection may, as is often clearly seen, be by direct contact, and it may be difficult to determine in some sections where the caseous bronchial gland terminates and the pulmonary tissue begins. In other instances it takes place along the root of the lung, and is subpleural. Among rarer sequences may be mentioned diverticula of the œsophagus following adhesion of an enlarged gland and its subsequent retraction, and, in the case of the anterior mediastinal and aortic groups, the frequent association of tuberculous adenopathy and pericarditis, either by contact or by rupture of a softened gland into the pericardium.

Symptoms.—In the great majority of instances there are no indications whatever, and even in enormous enlargement pressure-signs may not have been present. Authors differ extremely in their views on this point. Many hold, and I think correctly, that the manifestations, as a rule, are very slight. Compression of the veins leading to dropsy, dilatations of the veins causing cyanosis, and hæmorrhages are referred to by Barthez and Sanné. Alterations in the character of the heart-sounds and attacks of paroxysmal dyspnœa are described by the same writers. The latter come on suddenly, often at regular hours, frequently in the afternoon, and there is extreme oppression with rapid breathing, cyanosis, and cold sweats, almost like an attack of severe croup. These paroxysms may succeed each other, and they have been ascribed not so much to pressure at the bifurcation of the trachea as to compression of the vagi, causing in this way laryngeal spasm. More definite, undoubtedly, is the compression of one or other bronchus, causing feeble breathing on the side most

affected, with sibilant and fine râles. Usually, however, when the glands are very much enlarged the lung is also involved, and it may be difficult to say how far the alterations are due to the changes in it. Still less reliable is the information obtained on percussion, for the dullness in the upper part of the sternum and in the interscapular spaces is, when present, by no means a positive sign. The thymus may cause sternal flatness on percussion; and behind, unless the glands are enormously enlarged and the child very thin, it is difficult to determine any special modification of the resonance in the interscapular space between the first and third dorsal vertebræ.

(*d*) **MESENTERIC GLANDS (TABES MESENTERICA).**—The glands involved are those of the mesentery and the gastro-hepatic omentum and the chain of retroperitoneal glands along the aorta; more rarely those of the pelvis. Tuberculous disease of these glands is extremely common; thus of 127 cases of fatal tuberculosis in children, noted by Woodhead, these structures were involved in 100, while Ashby states that of 103 consecutive post-mortems on children dying of tuberculosis, in 62 there was tuberculous ulceration of the intestines; in 71, cheesy mesenteric glands; in 55, both ulcers and cheesy glands; in 7, tuberculous ulcers without involvement of the glands; and in 16, cheesy glands without ulcers. Of 144 children in which the mesenteric glands were tuberculous, only 44 showed neither ulcerations nor tubercles in the intestines (Barthez and Sanné).

In a great many instances the condition is found accidentally in children who have died of other diseases. Unquestionably, as is indicated by these figures, the infection in many of these cases is primary in the glands. Lesion of the intestines is not necessary. Some experiments have shown that the bacilli may gain entrance through a healthy mucosa. A special interest relates to the possibility of infection by the bacilli in milk, more particularly as it is well known that in animals experimentally fed with infected milk primary tuberculosis of the intestines, with extensive disease of the mesenteric glands, has been produced. The question will be referred to again on the subject of primary tuberculosis of the intestines. The cases fall into four groups:

(1) Very slight tuberculous affection of a few glands (which may be the only ones), met with accidentally in children who have died of various disorders.

(2) In the chronic generalized tuberculosis, in both the acute and chronic pulmonary tuberculosis, and in the more chronic forms of tuberculosis of any of the organs in children, the mesenteric glands may be found enlarged and caseous. There are instances, too, in which the affection of the mesenteric and retroperitoneal glands with those of the thorax constitutes the chief lesion.

In both these groups the disease of the glands does not necessarily cause any symptoms pointing to abdominal disorder.

(3) In a third group there are signs of chronic intestinal catarrh or ulceration and very marked disturbance in the general nutrition. These cases are seen chiefly in children between the ages of eighteen months and five years. The abdomen is distended, tympanitic, usually a little painful on deep pressure, but no nodules are felt. The diarrhœa is the most troublesome symptom; the stools are frequent, brownish or yellow-brown in color, containing mucus, not often blood. The diarrhœa is variable, and may sometimes persist for several weeks. There is usually slight fever, but the general wasting and debility are the most characteristic features. The name *tabes mesenterica* is often applied to this condition. The course is chronic and may extend over a

year or two, leading to the most extreme emaciation. It is sometimes very difficult to determine whether actual tuberculous disease of the bowel is present or not, as a chronic intestinal catarrh may lead to just such a condition of extreme debility and wasting. In the diagnosis of these cases much stress can be laid upon the presence or absence of tubercles in other parts.

(4) And, lastly, there are cases in which with ulceration of the intestines the mesenteric glands are greatly enlarged, and in addition the peritoneum is involved. Here the diarrhœa, the slight fever, the malnutrition, and progressive wasting are as in the previous group; additional symptoms are associated with disease of the peritoneum, in which nodular masses may be felt, and there may be considerable ascites. These cases will be referred to more particularly under Peritoneal Tuberculosis.

(2) TUBERCULOSIS OF THE INTESTINES AND OF THE ABDOMINAL ORGANS.

(a) TUBERCULOSIS OF THE BOWELS.—The small intestine is most frequently involved; thus, of 141 children presenting tuberculous ulcerations in the gastro-intestinal canal (Barthez and Sanné), in 134 the small intestine was involved; in 60, the large intestine; in 71, the small intestine alone. It is remarkable, considering the comparative rarity in the adult of tuberculous disease of the stomach, that in this series it should have been met with in 21 cases. That tuberculosis may originate in the alimentary canal is shown experimentally by the feeding of guinea-pigs with cultures of the bacillus and the feeding of calves and pigs with the milk of tuberculous animals. There are now many series of cases demonstrating the facility with which animals may be infected through this latter source. That the intestinal lesion may be primary in children is acknowledged. The comparatively large number of children with caseous foci in the mesenteric glands is very suggestive. On the other hand, instances of primary intestinal tuberculosis are not very common.

In a great majority of the cases the tuberculous lesions are part of a general infection, and are undoubtedly secondary. The ulcers are situated chiefly in the ileum, involving the solitary and agminated follicles of Peyer. The tubercles may be seen as small granulations in the submucosa; sometimes the whole ileum may present a remarkable appearance from the grayish-yellow nodular tubercles, the size of split peas, occupying the submucosa and the mucous membranes. The caseation and necrosis lead to ulceration, which may be very extensive, involving at first Peyer's patches, but ultimately extending beyond their limits. The tuberculous ulcer has the following characters: It is "transverse to the long axis, rarely ovoid, often irregular in outline; the edges and base are infiltrated, often caseous; the submucosa and muscularis are also involved in the tuberculous process; and, lastly, colonies of young tubercles or well-marked lymphangitis may be seen on the serosa."

Primary tuberculosis of the bowel is, as stated, rare; but in children with extensive ulceration in the ileum and very slight lesions of other parts the disease may be regarded as primary; thus in a child aged nine who was admitted to my wards with dropsy and emaciation after an illness of six months' duration, there were only a few small foci in the lungs, while the intestines showed most extensive disease. About 50 cm. below the duodenum there was a large circling ulcer, the edges of which were undermined, the bases irregular and worm-eaten, and containing necrotic, grayish material. The peritoneum over it was thick and opaque. Throughout the whole of the ileum there was a series of these girdling ulcers at varying intervals. The cæcum presented a very

large, deep ulcer, while the mesenteric attachment about the ileum formed a large tumor-mass from the extent of the involvement of the glands. The peritoneum presented scattered tubercles and the mesenteric glands were enormously enlarged.

In a few instances tuberculous disease of the bowels extends from a chronic tuberculous peritonitis in which the coils of the intestine become matted together, caseous and suppurating foci develop between the folds, and perforation may occur in several places.

Symptoms.—The symptoms of intestinal tuberculosis are very varied. The most common indication is a persistent diarrhœa. It is not always, however, proportionate to the extent of the ulceration, and large ulcers in the ileum may exist with constipation. When the ulceration is extensive in the large intestine the diarrhœa is usually profuse and obstinate. The mode of onset is variable. In a few instances of general tuberculosis there is diarrhœa from the start. In a large number of cases the existence of intestinal complication is not suspected until the signs of disease in other organs are well marked; and in perhaps a majority of the secondary cases the diarrhœa is rather an event of the latter part of the illness. Of other symptoms, hæmorrhage may occur, or peritonitis from extension—a condition not very uncommon, and often associated with disease of the mesenteric glands. The abdomen in these cases is usually enlarged and painful, and the nodular masses may be felt. In a few instances there are gastric symptoms, which do not necessarily indicate ulceration in the stomach, but there may be loss of appetite and occasional vomiting, and there are instances on record of profuse hæmatemesis or melæna from erosion of an artery.

The outlook is unfavorable, and death may be caused by the severity of the intestinal symptoms, or more rarely by the accidents, such as perforation or hæmorrhage.

Recognition is rarely difficult, except in the primary cases, which are regarded at first as simple entero-colitis. Usually, however, when well established, the diagnosis is easy, particularly when other organs become involved. In suspected cases the stools should be carefully examined for tubercle bacilli.

(*b*) **TUBERCULOSIS OF LIVER.**—In all cases of acute miliary tuberculosis granulations are found in this organ; sometimes they are extremely minute and are only detected microscopically. The liver is usually somewhat enlarged, pale, and fatty. In more chronic cases, particularly the diffuse generalized tuberculosis of young children, the tubercles may attain considerable size and develop about the finer bile-ducts. They undergo rapid softening, and give a very remarkable appearance to the liver, which is in extreme cases almost honeycombed with tuberculous abscesses, varying in size from a pea to a marble; the pus is usually bile-stained.

Occasionally large, coarse, caseous masses are found forming irregular tumors, most frequently in association with perihepatitis or tuberculous peritonitis. The so-called tuberculous cirrhosis of the liver does not, I believe, occur in children, though there may be in chronic cases of tuberculosis a marked increase in the connective tissue of the organ.

(*c*) **TUBERCULOUS PERITONITIS.**—Tuberculosis is one of the most common causes of peritonitis in children. It is more common about the eighth and tenth years, and attacks boys more frequently than girls; thus of 86 cases analyzed by Barthez and Sanné, there were from

1	to	2½ yrs.	11 cases.
3	to	5½ "	26 "
6	to	10½ "	40 "
11	to	15 "	9 "

The ratio of frequency in children may be gathered from the large statistics of Aldibert, who found in 326 cases of tuberculous peritonitis, 52 in children. As in the adult, the disease may be primary, but in a majority of the cases it is secondary to tuberculosis of the intestines, mesenteric glands, or of the genitalia.

Morbid Anatomy.—Tubercles in the peritoneum are not infrequently met with in the bodies of children dead of tuberculosis. Ashby noted them 38 times in 105 post-mortems on tuberculous children. They occur either as (1) the gray granulations with or without exudation, serous or sero-fibrinous. Sometimes the entire peritoneum is found studded with (2) firm, hard, fibrinous tubercles surrounded by a pigmented and firm connective tissue. In both of these varieties the process may be latent, and the condition is met with accidentally post-mortem. More frequently (3) when symptoms have been present, the tubercles are in the form of caseous nodules, yellow-gray in color, often forming flattened tuberculous plaques. The exudate is purulent or sero-purulent, the coils of intestines are much matted together, and between them there may be large caseous masses. It may be impossible to separate the coils, and in advanced cases extensive ulceration occurs, with multiple perforation of the intestine. There are three anatomical points of special interest in these cases: First, the effusion may be sacculated and form a definite tumor; sometimes the process is confined to the cavity of the lesser peritoneum; in other cases it is in the pelvis, less frequently in the middle portion. The cysts may be multi- or mono-locular.

Second, there are cases in which occlusion of the intestine has resulted, sometimes from compression of the coils by the large caseous masses; more frequently by the bands of connective tissue in the healing of the process. Aldibert has found five instances of this sort in children.

Lastly—and much more frequently in children than in adults—there is periumbilical suppuration. The intensity of the inflammation is in the central portion of the abdominal cavity, adhesions take place, and a definite cyst is formed, usually purulent, which projects at the umbilicus, and often opens spontaneously, leaving a fistula, sometimes stercoral, which persists for months but may ultimately heal.

Symptoms.—The symptoms of tuberculous peritonitis are extremely varied, and it is very difficult to give a clear and definite picture of the disease. For convenience three clinical types may be considered:

(1) *The Ascitic Form.*—The symptoms may come on acutely with a diffuse eruption of miliary tubercles. So abrupt is the onset that cases have been mistaken for acute enteritis, or even for acute obstruction or hernia. More frequently the onset is subacute, and ascites gradually develops. Fever of some degree, indigestion, and diarrhoea are present, and there may be abdominal pain; but in many instances the process is latent, and the enlarging abdomen is the symptom for which the physician is consulted. The effusion, indeed, may proceed to considerable degree without fever, and with no symptoms other than those of gradually-failing health and progressive emaciation. Intestinal disorder occurs in some instances, diarrhoea, colicky pains, or often attacks of diarrhoea alternating with constipation. The local symptoms are by no means characteristic. The abdomen is distended, the skin thin, the superficial veins

enlarged. Percussion gives dulness in the flanks, which is movable, resonance in the umbilical region, and there is a well-marked fluctuation wave. Palpation may be entirely negative; no nodular masses are felt. The liver and spleen are not often enlarged. It may be extremely difficult, or quite impossible, unless there are tuberculous lesions in other regions, to speak definitely of the nature of the gradually-developing ascites. The clinical picture is very similar, indeed, to that of the cases of ascites from cirrhosis, and an identical condition is met with in the rare cases of simple chronic peritonitis in children. The ascites may demand tapping, but the fluid reaccumulates rapidly. The exudate may be encysted, forming a prominent tumor in the epigastric or umbilical regions (in which case the effusion is probably within the lesser peritoneum), or it may be situated in the pelvis or in the flank, and simulate very closely cystic ovarian disease. This form is not very uncommon in children, and very good results have followed operation; of nine instances in the literature, all recovered. This ascitic form, developing slowly, and ultimately presenting the picture of a chronic ascites or an encapsulated exudate, is by far the most favorable variety, and cases may recover spontaneously or after operation.

(2) *The ulcerative form* is much more serious. The peritoneum here contains larger caseous masses which break down, and there is a diffuse purulent peritonitis. The coils of intestines are matted together, nodular tuberculous masses develop on the parietal and visceral layers, the glands are greatly enlarged, and in protracted cases extensive ulcerations occur. The onset in this form is usually gradual, but the abdominal symptoms are pronounced. The child complains of colicky pains, diarrhoea, and chronic indigestion. The abdomen is enlarged and painful. The condition on examination may be entirely different from that of the ascitic form. The outline is often symmetrical, not flattened in the flanks; nodular projections may sometimes be seen beneath the skin. Unless there is a very extensive purulent effusion there is no movable dulness. There is a flat tympany or there are alternating areas of resonance and dulness. On palpation there is a boggy, doughy feel, and nodular masses may be felt in different regions. The liver and spleen may both be enlarged. In this suppurative form the effusion may be general, or it may be encysted either in the upper abdominal region or in the pelvis. One form of this encysted suppurative variety requires special consideration—namely:

Periumbilical Tuberculous Abscess.—This is seen most frequently in children, and is in reality a localized suppurative peritonitis, which points at the navel and frequently opens and discharges. The condition is almost constantly tuberculous in the child. There may be a fistula discharging pus for weeks or months, and recovery may ultimately take place. In other instances the fistula communicates with the bowel. In the case of a colored child, aged five, operated upon by my colleague, Dr. Halsted, there was distention of the abdomen, marked protrusion of the umbilicus, and here a spontaneous opening discharging yellowish material for months. Then the opening healed and the condition of the child improved. At the time of the operation there was a large, prominent, cone-shaped, umbilical tumor. The child died some time after the operation; creamy pus was found between the intestinal coils, and there were many tuberculous ulcers in the intestines. There was an extensive caseous salpingitis.

There are instances also of perihepatic tuberculous abscesses.

(3) *Chronic Adhesive or Dry Tuberculous Peritonitis.*—In a very considerable number of all cases of tuberculous peritonitis there is little or no serous or purulent exudate, but the tubercles are surrounded with a fibrinous lymph

and they tend rapidly to cicatrize. The growing tubercles may not have caused any symptoms, and the condition is found accidentally post-mortem, and in adults has often been met with in exploratory laparotomies for various conditions. In long-standing cases the tubercles are hard, firm, often surrounded by deeply pigmented fibroid adhesions. In some of these instances the tuberculosis of the peritoneum is localized; thus it has been found in a hernial sac alone, or in the region of the cæcum and appendix, or on the epiploön. There are instances in which this membrane has been gradually curled and rolled until it forms a ridge-like tumor lying across the upper portion of the abdomen. This chronic adhesive form is not so frequent in children as in adults. The symptoms are very indefinite. The abdomen is usually distended and tympanitic, everywhere resonant, sometimes distinctly painful on pressure. In protracted cases the omentum may be felt as a firm ridge in the upper portion of the abdomen. The general symptoms are very variable. There may be wasting and cachexia, sometimes with marked fever, though these chronic adhesive forms are not infrequently afebrile throughout, or the temperature, indeed, may be subnormal. With the exception of the colicky pains there may be no symptoms directly from the peritoneum, but the cases are very often complicated with tubercles in other parts, and the mesenteric glands or the lungs may be extensively diseased. These are cases in which spontaneous recovery is not infrequent.

Diagnosis.—A gradually developing ascites in a young child with moderate fever is in itself very suggestive of peritoneal tuberculosis. Doubtless very many of the cases of simple ascites with recovery belong to this disease.

The condition is to be distinguished from ascites due to disease of the liver and from chronic simple peritonitis. Cirrhosis of the liver, syphilitic or simple, is a rare disease in children. The local symptoms may give us no clue, but after withdrawal of the fluid the liver in a cirrhotic case may be felt to be unusually hard, and perhaps small, and possibly, when due to syphilis, irregular. The general symptoms are more important. In cirrhosis there is more frequently a slight jaundice. The fever and gastro-intestinal symptoms are not so marked. An encysted exudate is always in favor of tuberculosis. A simple chronic peritonitis, though rare, occurs in children, and, even after the exploratory laparotomy, the diagnosis may not be clear, inasmuch as there may be small nodular fibroid bodies scattered over the membranes. It is very important in these cases to have a careful microscopical examination made, in order to determine the presence of bacilli, or, if the nodules are very firm and fibroid, the experimental test should be made. It is quite possible that some instances of reported recovery in peritoneal tuberculosis after laparotomy may have been instances of this chronic simple peritonitis with fibroid nodules. The ulcerative form with suppuration and the development of nodular masses in the peritoneum with fever and a marked cachexia, rarely offers the slightest difficulty in diagnosis. It is to be remembered, of course, that the suppurative forms also may be encysted, and the periumbilical abscess with umbilical fistula, simple or stercoral, is almost constantly tuberculous.

Prognosis.—The prognosis is often good, particularly in the ascitic and chronic adhesive varieties. Many instances, no doubt, in which the ascites has gradually disappeared have been tuberculous, and even in the ulcerative variety, when the abscess has discharged at the navel, recovery has followed. The operation of incision and drainage has certainly favored recovery in a considerable number of cases.

Treatment.—The general treatment of tuberculosis will be discussed at the end of the section; here reference will be made more particularly to incis-

ion and drainage in tuberculous peritonitis. The results which have been obtained are exceedingly satisfactory, even if we suppose, as is probable, that many cases relapse and are not fully healed at the time of reporting. The figures given in the monograph of Aldibert are extremely interesting: in the ascitic form, of 32 instances in which laparotomy was performed, there were 3 deaths and 29 recoveries, 4 of which had persisted for more than one year. This demonstrates the impunity with which the abdominal cavity may be opened, and the large percentage, at any rate, of those which are benefited immediately by the operation. In the chronic adhesive form an operation is really not indicated, as in the majority of the instances the tuberculosis is in process of healing, but there are cases in which pain, associated with the adhesions, has been relieved by an exploratory incision. In the ulcerative variety, when generalized, the results have not been so satisfactory, but many instances with an encysted purulent fluid have been opened and drained successfully. The drainage favors the process of cicatrization in the tubercle, lessens the tendency to effusion, and exerts a favorable influence on the whole process. Of the 52 cases in children in which laparotomy was performed, there were 45 recoveries and 7 deaths. Of these 45, 9 had persisted for more than a year, and 2 for more than two years (Aldibert).

(3) TUBERCULOSIS OF THE LUNGS.

In speaking of acute miliary tuberculosis and of chronic diffuse tuberculosis we have considered affections in which the lungs are almost constantly involved—in the one case the seat of miliary granules; in the other of larger, coarse, grayish-yellow tubercles. We shall speak in this section more particularly of those forms in which the lungs are so involved, that the clinical features are those of an acute or of a chronic pulmonary disease. Two groups of cases may be recognized: the acute tuberculous broncho-pneumonia, and the chronic ulcerative form, the first corresponding to the acute galloping phthisis, and the other to the chronic phthisis, or, as we call it now, chronic pulmonary tuberculosis.

(a) ACUTE TUBERCULOUS BRONCHO-PNEUMONIA.—In infants and children we very rarely see pulmonary tuberculosis set in with the clinical picture of an acute lobar pneumonia. Personally, I never remember to have met with an instance, such as is not very rare in adults, in which the tuberculosis came on abruptly, and at first ran the course of an ordinary lobar pneumonia, with pain in the side, high fever, and rapid consolidation of an entire lobe. Such cases are, however, on record, and it is only the absence of the crisis, the persistence of the local signs, the gradual softening, and the development of hectic and progressive debility which lead to a revision of the diagnosis. It is to be remembered that while clinically the physical signs may be those of a lobar affection, anatomically it is clearly seen that many groups of lobules are involved, separated by strands of air-containing or collapsed lung-tissue. These *pseudo-lobar* cases are almost impossible to differentiate during life.

Tuberculous broncho-pneumonia is common in children from the sixth month to the fifth year. A large proportion of the cases occur after the second year.

The disease is most common in children in institutions, in those debilitated by previous illnesses, and more particularly in convalescents from one of the infectious diseases—measles, whooping-cough, scarlet fever, or diphtheria. It is most frequent perhaps after measles and whooping-cough. Its sequence in the latter disease has been common knowledge in the profession since the days

of Willis, whose axiom, "*Tussis convulsiva vestibulum tabis*," has been quoted through two centuries. Children the subject of chronic naso-pharyngeal catarrh and tonsillitis, and mouth-breathers seem more prone to the affection. But it is to be remembered that it may develop in perfectly healthy, well-nourished children.

And lastly, like miliary tuberculosis, it may be a terminal process in cases in which local tuberculous disease exists in other parts—the skin, bones, lymph-glands, or the urogenital tract.

Morbid Anatomy.—The condition varies considerably with the intensity and duration of the process. The lungs may be voluminous and crepitant, with firm and nodular masses scattered throughout the lobes. On section these are seen to be peribronchial nodules ranging in size from a pea to a walnut. Some of the more recent are reddish in color; the older are grayish-yellow, with, perhaps, central softening. Many of these peribronchial nodules are seen to be composed of aggregations of tubercles undergoing caseation. In the very acute cases the process is more extensive in the upper lobes or central portion of the lungs, certain parts of which may be almost solid and scarcely contain any air. The consolidation may indeed look uniform, but on section it is noted that the process is not actually diffuse, as in a lobar pneumonia, but the general consolidation has arisen from the involvement of a very large number of the lobules, groups of which are separated by strands of reddish collapsed tissue. The consolidated areas have undergone caseation, and may in places have softened, forming cavities. The older the process the more extensive usually are the areas of caseation. Though primarily tuberculous, many of these cases show a mixed infection, and there may be areas of simple broncho-pneumonia due to streptococci, staphylococci, or pneumococci. The pleura may show many nodules or a fresh, fibrinous exudate, sometimes a sero-fibrinous or even purulent exudate. The bronchial and tracheal glands are enlarged, tumefied, and studded with tubercles or uniformly caseous, not infrequently having softened to form definite abscess. The glands at the hilus may be greatly enlarged and extend deeply between the lobes, and in some instances there would appear even to be an invasion of the lung-tissue from these deeply-placed large caseous glands. The other organs may present a few scattered tubercles or there may be a generalized miliary tuberculosis.

As in other forms of broncho-pneumonia, the essential lesion is a bronchitis and peribronchitis excited by the tubercle bacilli, with inflammation of the contiguous air-cells, which become filled with epithelial products, the so-called catarrhal alveolitis. The accompanying phenomena of atelectasis and emphysema occur just as in simple broncho-pneumonia, and the distinguishing features are the caseation and necrosis with the presence of the bacilli.

Much discussion has taken place upon the relation of broncho-pneumonia to tuberculosis, and some French observers have maintained that in many instances the form following measles and diphtheria, and which anatomically looks simple in character, is in reality tuberculous and due to the bacilli. It may be difficult sometimes to determine whether a given patch of broncho-pneumonia is tuberculous or not, but as a rule, macroscopically, there will be seen small tubercles or areas of caseation, while in stained sections the bacilli are readily demonstrable. The simple broncho-pneumonia in some cases precedes the tuberculous, particularly after measles, scarlet fever, diphtheria, and whooping-cough. In institutions it is by no means uncommon to meet with cases in which broncho-pneumonia has gradually subsided, and then symptoms have developed pointing to fresh invasion, and ultimately death follows with the lesions of an acute, recent, tuberculous broncho-pneumonia. Sometimes the

infection is less intense, and a subacute or chronic pulmonary tuberculosis is established. In cases of tuberculosis consecutive to broncho-pneumonia we find the lesions of two sorts: simple, inflammatory, non-tuberculous, such as peribronchial suppuration, dilatation of the bronchi, lesions of the alveolar epithelium, and peribronchial and peri-alveolar sclerosis; then, in addition, there are the true tuberculous processes, peribronchial nodules, tuberculous infiltration, and caseous areas (Mosny).

In other instances the tuberculosis precedes the broncho-pneumonia. This is met with particularly in children the subject of latent tuberculosis, in whom, following one of the infectious diseases, a simple broncho-pneumonia develops. According to Mosny, the lesions may be seen as an alveolitis surrounding the tuberculous peribronchial nodules, or foci of simple and tuberculous broncho-pneumonia occur scattered throughout the apices of the lung. It is a broncho-pneumonia dependent upon pneumococci or streptococci invading a lung already the seat of local tuberculosis.

Symptoms.—Clinically, tuberculous broncho-pneumonia scarcely differs in any feature from the simple form. The onset may be acute in a previously healthy child, but more frequently the disease sets in during convalescence from one of the infectious diseases. In the tuberculous form the fever is sometimes not so high and not so persistent, showing more variations throughout the day. Cough and dyspnoea are prominent symptoms. The physical signs are those of broncho-pneumonia. The localization of the lesion is more commonly at the apices of the lung, where there may be signs of consolidation with fine crepitant and subcrepitant râles. There are no physical signs of any moment in differentiating a simple from a tuberculous broncho-pneumonia, and indeed even the localization of the disease at the apex, upon which so much stress is laid, is not of very much value, since we frequently find in young children a tuberculous process beginning at the base or in the central portions of the lung. In the course of the disease, however, indications of great value develop; thus toward the end of the second week there are more marked oscillations in temperature, often with profuse sweats. The child emaciates rapidly, and there may sometimes develop signs indicating softening. In the acute cases the duration is from three to five weeks. Throughout the course of the disease there may be no single indication of much value in definitely determining the nature, and we often have to depend more on the general features of the case. Careful inquiries should be made as to heredity; also the personal history immediately preceding the onset. Sometimes important information may be gathered by a systematic examination of the child. There may be a tuberculous adenitis, local bone disease, or a tuberculous testis. Simple broncho-pneumonia tends as a rule to recovery; in exceptional cases, however, it becomes subacute, and ultimately chronic. In the more subacute and chronic cases tuberculous broncho-pneumonia may present large areas of caseation, which give the physical signs of consolidation, perhaps of an entire lobe. In such instances softening and the signs of cavity not infrequently develop, and give very definite indications of the nature of the process. As the little patients rarely expectorate, examination for bacilli can seldom be made. Sometimes, if vomiting occurs, portions of mucus may be picked out, and important evidence in this way obtained.

(b) **CHRONIC PULMONARY TUBERCULOSIS.**—In infants and very young children we find the lungs either involved in a generalized tuberculosis or the seat of an acute tuberculous broncho-pneumonia. After the sixth or eighth

year cases are not very uncommon in which the picture resembles that of chronic tuberculosis pulmonum of the adult.

Morbid Anatomy.—The lesions are similar to those met with in the tuberculosis of adults—miliary tubercles, peribronchial nodules, caseous blocks, areas of softening and of fibroid induration, and cavities of various sizes. We do not see so frequently the invasion of the lung from the apex downward. The chief seat of disease may be in the central portion of the lung, or even at the base. As already mentioned in speaking of tuberculosis of the lymph-glands, the groups along the trachea and about the bronchi may be greatly enlarged and caseous, forming on section a very striking feature in the chronic pulmonary tuberculosis of children. Indeed, in some instances the process seems to spread directly from the deeply-placed glands in the hilus of the lung, which may be enormously enlarged, uniformly caseous, and the organ may be directly invaded from them. Large areas of caseous pneumonia are not uncommon, and often present foci of softening. Small cavities are by no means infrequent in chronic pulmonary tuberculosis of children, but very large excavations are rare; thus in the 265 cases noted by Barthez and Sanné there were 77 cases with excavation, chiefly, too, in the upper lobes. In the analysis by Leroux of the cases of the late Professor Parrot, in 219 children under two years of age there were 57 instances in which cavities existed. In 5 of these the children were under three months. In long-standing cases hard, firm, fibrous tubercles are found, and sometimes cretaceous nodules. The primary lesion in a great majority of instances is a tuberculous broncho-pneumonia, taking its origin in the smaller bronchioles, leading to peribronchial nodules and subsequent peribronchial alveolitis.

Symptoms.—The general symptomatology of chronic pulmonary tuberculosis in the child is similar in essential details to that of the adult, but presents, however, as might be expected, certain peculiarities. The onset is generally more abrupt, and the first symptoms may be those of a broncho-pneumonia at the apex. The child may have been in failing health or come of a markedly tuberculous stock, or there may have been local glandular or bone disease. Occasionally failing health, with repeated attacks of chills and fever, may arouse the suspicion of malaria, but this mode of onset is not so frequent as in adults. Some cases follow a protracted naso-pharyngeal catarrh with recurring bronchitis. Progressive failure in health and strength, cough and fever, are the first symptoms to attract attention. There is loss of appetite, but rarely the extreme anorexia which we find in some cases of pulmonary tuberculosis in older subjects. Cough is rarely absent among the initial symptoms, and, with variations, persists. It is short and dry at first, subsequently looser. It may be distributed equally throughout the day or is most troublesome at night, and paroxysms of coughing may return at fixed hours, so that the case may be mistaken at first for whooping-cough; but there is never the noisy crowing inspiration. Expectoration is absent in very young children. Children above the age of ten can often be taught to expectorate. The sputum is mucoid at first, with grayish-yellow streaks; sometimes it is more sero-mucoid, and in the later stages more definitely purulent. Hæmoptysis may be said to be infrequent in children under ten. Certainly it is very rare at the onset. It is usually small in amount. The terminal hæmoptysis, common in the adult, but rare in children, results from the rupture of an aneurism in a small cavity or erosion of a branch of the pulmonary artery. The fever of onset and during the early periods is remittent, the daily excursions slight—a range between 102° and 104° is common. Subsequently, when the disease is more extensive and softening has taken place with the formation of cavities, the temperature is more

hectic in character, and the morning observation may be normal or subnormal, while in the evening the thermometer may register 103.5° or 104° , or even higher. Chills are not very common. Drenching sweats are frequent, particularly toward the close. Dyspnoea may be present at the onset and during the early stages, and may be due in part to the fever, sometimes to the presence of a diffuse bronchitis. Marked increase in the respirations, with cyanosis, indicates very rapid progress in the disease. In protracted cases, just as in the adult, there may be very extensive destruction of the lung without the slightest dyspnoea. The child may complain of pains in the chest, usually associated with pleurisy. In a majority of instances the disease is painless throughout its course. Qvisling states that an early sign is tenderness on percussion of the affected side, or on pressure in the intercostal spaces, particularly in the first space at the apex.

Progressive weakness and wasting are very pronounced symptoms, and there is usually progressive pallor. Frequently the abdominal viscera become involved, and there is diarrhoea due to tuberculous ulceration, and the liver and spleen may become enlarged. The urine does not often show changes, but as the disease progresses albumin is common and a secondary nephritis may develop. A child may come under observation with general anasarca, due partly to the anæmia, partly to the renal condition, and the pulmonary tuberculosis may be entirely overlooked.

Physical Signs.—*Inspection* frequently shows in advanced cases an extremely thin chest, with marked intercostal spaces. Deformities due to mouth-breathing or to rickets are not uncommon. On the affected side the respiratory movement may be decidedly less marked, or the clavicle may stand out prominently; or there may be subclavicular depression at the affected apex—a sign usually of a chronic process. In very long-standing cases with much fibroid change there may be flattening of the affected side, with depression of the shoulder.

By *palpation* one appreciates any differences in expansion on the two sides, and the differences in the tactile fremitus, and it may be of value in eliciting painful points.

Percussion.—In the early condition, when the tubercles are scattered or the areas of broncho-pneumonia are limited, there may be no change in the percussion note. Indeed, the emphysema about the affected areas may cause slight hyper-resonance over the part affected. Extensive involvement at one apex usually gives loss of resonance beneath the clavicle, which may amount to dullness and is accompanied with marked increase in the resistance. Absolute flatness is rarely met with. Skoda's resonance, the flat tympany, is not frequent. The cracked-pot sound has very little value in children, as it may sometimes be elicited in a thin-walled healthy subject.

Auscultation may give only the signs of bronchial catarrh, piping râles and moist sounds, but when there is definite dullness there is usually change in the character of the respiratory sounds, which have lost their vesicular character and are harsh, broncho-vesicular, or definitely bronchial. Sometimes with defective resonance there is enfeeblement of the respiratory murmur, with prolongation of expiration. The auscultatory phenomena are often very deceptive. Diffuse bronchitis may lead us to suppose that there is much greater involvement of the lung than in reality exists. In very young infants signs of cavity are rarely present, but in older children in advanced cases, with hectic and emaciation, the metallic splashing or amphoric quality of the râles, with the loud cavernous breath-sounds, leaves no doubt as to the existence of a vomica. In children, more frequently than in adults, we are deceived by the

so-called pseudo-cavernous signs. Over an area of slightly defective resonance or of positive dullness inspiration and expiration are cavernous, the râles large and resonant, and the whispered voice may be conveyed intensely to the ear. In acute cases with high fever one is not so apt to be deceived; these signs are also met with in broncho-pneumonia and in pleurisies.

Course.—The course of chronic pulmonary tuberculosis is more rapid in children than in adults, and a majority of cases die in from six to twelve months. The disease is marked, now by intervals of improvement, in which the fever lessens and the severity of the symptoms subsides, now by aggravation of the local and constitutional condition, sometimes with attacks in which the fever and dyspnœa increase, and the child may become quite cyanotic. Some of these intercurrent attacks simulate closely acute tuberculosis, but often pass away at the end of a week or ten days. In the chronic cases they probably indicate the invasion of other portions of the lung.

Occasionally, in a case of chronic pulmonary tuberculosis extensive fibroid substitution takes place, with gradual retraction of the affected side, depression of the shoulder, and all the signs of so-called fibroid phthisis. Usually in such instances there is dullness at the base and side with modified resonance, and cavernous signs at the apex. When involving the left lung, the heart is drawn over, and there may be a very extensive cardiac pulsation from the second to the fifth interspaces. A child may gradually regain a fair measure of health and for years live a tolerably comfortable life, troubled only by one or two spells of coughing through the day. There may be dyspnœa on exertion, and gradually the terminal phalanges become clubbed. Hæmoptysis is rare, but occasionally terminates the case.

Diagnosis.—Progressive emaciation with hectic and cough in a child should always arouse the suspicion of chronic pulmonary tuberculosis. In the early stages the condition is usually that of tuberculous broncho-pneumonia. Careful and repeated physical examination may be necessary to establish the diagnosis, and one should take into consideration carefully the condition of the other organs. The position of the physical signs at the apex or central portions of the lung, the increased fremitus, the moist sounds, are all suggestive, and frequently one may trace the progressive character of the lesion. The disease most frequently confounded is empyema, but here the movable dullness, the bulging of the intercostal spaces, and the absence of fremitus are valuable points.

Auscultation is an extremely fallacious guide, and in several instances the persistence of a loud, almost cavernous, respiratory murmur at the base has led the practitioner astray. When in doubt the exploratory needle should be freely used for the purpose of diagnosis. The differentiation of chronic simple broncho-pneumonia sometimes gives a great deal of trouble, and the time element alone may determine whether we have to do with a tuberculous process or not. These are the very instances in which any fragments of sputum should be carefully sought for and examined. In a paroxysm of coughing the child may bring up a mouthful of food, and with it the expectoration, which should be carefully picked out and examined for tubercle bacilli.

Prognosis.—The prognosis in a large majority of the cases is bad, particularly when hectic is established and there is disorganization of one lung. On the other hand, when cases are seen early and placed under suitable conditions recovery may take place. The large number of individuals whose lungs and bronchial glands present traces of old tuberculous processes shows how considerable a proportion of all those who are infected must survive. We do not see many cases of chronic pulmonary tuberculosis in children between the ages

of six and fifteen, for the reason, no doubt, that the tuberculous broncho-pneumonia is so often an acute process, carrying off the victim before it has assumed the characters of a chronic affection.

(4) TUBERCULOSIS OF THE PLEURA.

This is usually secondary to existing disease in the lung or in the bronchial glands. A certain number of acute serofibrinous pleurisies in children may be, as in the adult, due to tuberculosis; but the cases, as a rule, run a favorable course, and unless the child has definite manifestations of tuberculosis in other parts the assumption in any given case is of course purely gratuitous. Purulent pleurisies in children are most commonly associated with lobar or broncho-pneumonia, but in a certain proportion of the cases the process is tuberculous. The disease is usually latent, and failing health, pallor, and shortness of breath are the symptoms for which relief is sought. The general symptomatology and diagnosis of tuberculous pleurisy are practically those of the simple forms which are elsewhere considered.

(5) TUBERCULOUS PERICARDITIS.

This is by no means rare in children, and cases have been reported in infants under a year. In 65 cases collected from the literature by Brackman, 19 were in children. The disease is associated in almost all instances with tuberculosis of the mediastinal or bronchial glands. An enlarged and softened gland may perforate the pericardium and produce an acute sero-fibrinous or suppurative inflammation; and no doubt a considerable number of all the cases of so-called idiopathic suppurative pericarditis have been due to this cause. The tuberculous process may slowly invade the pericardium from the mediastinal glands, and produce a chronic adhesive pericarditis, leading to great thickening of the membranes and gradual hypertrophy of the heart. The patient may die with all the symptoms of cardiac dropsy.

(6) URO-GENITAL TUBERCULOSIS.

(a) TUBERCULOSIS OF THE KIDNEYS.—As part of a general diffuse tuberculosis these organs are very frequently affected—more commonly, indeed, than in adults. Usually there are scattered gray tubercles or coarse yellow nodules in the cortical substance. Sometimes, however, the lesion is primary, and one or other kidney is extensively diseased. The affection in these cases appears to begin in the papillæ and calices, gradually invades the substance, and may ultimately destroy the entire organ, converting it into a series of excavations containing a cheesy material. When confined to one kidney, this (known as the *scrofulous* kidney) is sometimes met with in children, the other kidney being healthy and greatly enlarged. When there is extensive tuberculous pyelo-nephritis there is often pain over the kidney; the urine contains pus, very rarely blood. Irregular fever and chills are common. Frequent micturition may lead to the diagnosis of cystitis, with which, of course, it is frequently associated; but it is to be borne in mind that in connection with either calculous or tuberculous pyelitis frequent micturition may be a marked symptom. Sometimes the tuberculous organ is large enough in a child to be palpable. Tuberculosis rarely produces so extensive pyonephrosis as that due to stone.

The diagnosis can rarely be made from calculous pyelo-nephritis except by the detection of bacilli in the urine.

Tuberculosis of the ureters and bladder, very rare as a primary affection, is nearly always secondary to disease of the pelvis of the kidney, sometimes to disease of the prostate.

(b) TUBERCULOSIS OF THE TESTIS.—Disseminated miliary tubercles may be present in the testicles, but primary tuberculosis of these organs is not at all rare in children. Dreschfeld has reported an instance of congenital tuberculosis of the testis. Many cases have been reported of late years. Of 20 cases by Jullien, 6 were under one year, and 6 between one and two years. Both organs may be affected. The disease most commonly develops in the tunica albuginea or in the epididymis, and may lead to the formation of hard circumscribed tumors. In other instances the process may be more diffuse. When the nodular masses are large the testis may have a dumb-bell or double outline from enlargement of the epididymis. It is a serious affection in children, usually associated with tuberculous disease in other parts. Its existence should always be borne in mind, as in obscure abdominal or thoracic affections the presence of nodular masses in the testicles is of great help in diagnosis. The lesion may gradually heal. The cheesy masses may break down and suppurate, and, forming adhesions to the skin, the pus discharges, and the organ may become much enlarged—the condition formerly known as strumous orchitis.

(c) TUBERCULOSIS OF THE FALLOPIAN TUBES, OVARIES, AND UTERUS.—These parts are rarely affected primarily in children. It is not very uncommon in generalized tuberculosis to find, even in infants, a double salpingitis.

IV. PROPHYLAXIS.

While the possibility of inherited transmission from an infected mother cannot be denied, we have to face the fact that in a large proportion of all cases of tuberculosis the infection is at the gateways of the body—namely, in the bronchial and mesenteric lymph-glands—and we have here a clue to the two chief sources of danger.

To ensure freedom from contamination through the air the greatest care should be taken to prevent tuberculous patients spitting about in a careless manner. Every part of the expectoration should be carefully collected and boiled, and the patient's handkerchiefs should be thrown into boiling water. The liability of children to infection from this source is very much greater than that of adults, possibly on account of the intimate relations which the child has to the members of the family, more particularly the mother should she happen to be diseased. The habit of young infants, as they creep about, of putting everything in their mouths enhances greatly the liability to contamination.

The second danger to be avoided in children is the use of milk from tuberculous animals. Experiments have shown the readiness with which young pigs and calves become infected when fed on the milk of tuberculous cows. We have, unfortunately, no reason to believe that children are less susceptible than calves. Fortunately, the health authorities have at last awakened to the importance of careful inspection of dairy herds. The safeguard lies in the use of boiled milk, unless the source is known to be free from all possibility of contamination. The infection through meat is probably a very slight danger in a community.

Individual prophylaxis is of almost equal importance. A child born of delicate parents or in a family in which tuberculosis has prevailed should be reared with the greatest care. Very special pains should be taken to guard it against catarrhal affections of all sorts, particularly of the nose and throat, and

on the first indication of mouth-breathing a thorough examination of the nasopharynx should be made and any adenoid vegetations removed; and if the tonsils are at all enlarged, it is better to have them cut out. The child should live in the open air as much as possible, and the nursery should be thoroughly ventilated, more particularly at night. The meals should be at regular hours, the food plain and nutritious. Every encouragement should be given to take fats, and milk and cream should be used freely. It is a good practice for the mother to sponge the throat and neck of the child night and morning with cold water.

The trifling ailments should be carefully watched. The convalescence from measles, scarlet fever, diphtheria, and whooping-cough should be specially guarded. As the child grows older a systematically regulated exercise or course of pulmonary gymnastics may be taken.

V. TREATMENT.

Fortunately, a very large proportion of all cases of tuberculosis recover. Many instances of adenitis and disease of the bones heal spontaneously. Even in pulmonary tuberculosis it is remarkable how often we find post-mortem evidences of healed lesions, the percentage in some series being as high as 38. In fact, one may say that in a very large number of all cases in which the bacilli find a lodgment in the glands and in the solid organs, the conditions not being favorable, the growth remains local and tends to heal spontaneously. The essential point in the treatment of tuberculosis is the maintenance of nutrition at the highest possible grade. To aid in this three measures are to be practised:

First: A life in the fresh air and sunshine. The importance of environment is well shown in Trudeau's experiments with inoculated rabbits. Those confined in a damp, dark place succumbed rapidly; those allowed to run wild recovered or showed very slight lesions. By far the most important single element in the treatment of tuberculosis of all forms is the constant inhalation of fresh air. The good effects obtained at Göbersdorf, Falkenstein, Saranac Lake, Davos, and Colorado are due primarily to the fact that the patients live a life in the open air and sunshine. Even in cities much can be done by insisting upon open windows night and day, except, of course, in the very inclement seasons. It is an easy matter to protect the patient from draughts, and neither fever, cough, nor night-sweats contraindicate in any way fresh air. This is in reality the very essence of the climatic treatment of tuberculosis; that other considerations, such as moisture, barometric pressure, temperature, etc., are secondary is well shown by the fact that cases of various types of tuberculosis recover completely at places so diametrically opposite as Colorado Springs and Torquay. The regions of high altitudes with low barometric pressure are certainly more stimulating, and, according to Jaccoud, are better for cases of early pulmonary tuberculosis. Cases of bone and gland tuberculosis do remarkably well at the Adirondacks and in Colorado. The level regions with low barometric pressure, such as Riviera, Florida, and Southern California, are reputed to be more sedative in their action and better for tuberculosis in the more advanced grades and with high fever.

The second important measure is feeding, and the outlook in any case, particularly of pulmonary tuberculosis, depends very much upon the stability of the digestive powers. In no way does the open-air treatment do more good than in improving the appetite and digestion. A highly nitrogenized diet, consisting of broths, eggs, milk, and meat, should be taken. In children the milk

diet is particularly to be commended while fever persists. Raw meats scraped, various meat extracts, and peptones may be used when the digestion is feeble. In tuberculous children it is sometimes extremely difficult to manage the diet, and many patients have an aversion to the very articles of food which seem best adapted. Gavage can rarely be resorted to with any advantage in them.

Third, the use of such remedies as cod-liver oil, hypophosphites, and arsenic, which improve the general nutrition. Other measures are frictions, rubbing, and bathing, all of which stimulate and improve the general metabolism.

Treatment directed to the Tuberculous Processes.—The specific treatment by the tuberculin of Koch, which consists of a glycerine extract of the cultures of tubercle bacilli, has been practically abandoned, though the good results obtained in the hands of Trudeau and others with Hunter's modification raise the hope that something yet may be accomplished by its use. Antibacillary medication is as yet unknown, and the introduction of various antiseptic agents by inhalation, subcutaneously, or directly into the local lesion has not been followed by very brilliant results. The direct action of iodoform on local tuberculosis is of great interest, and the remarkable effects in joint tuberculosis should encourage a more widespread use in other forms of the disease. Creasote is a remedy which is believed to have a beneficial action on the tuberculous processes. It probably has no definite antibacillary action, though it is stated to influence powerfully the secondary and associated infections so common in tuberculosis. It seems rather to act as a general nutritive stimulant, improving the appetite, diminishing the fever, and promoting tissue-metabolism and, according to some, sclerotic processes. It is probably at present more widely used than any other single remedy. It has been a favorite with some practitioners for many years, and its reintroduction has been due to the powerful advocacy of Sommerbrodt, Bouchard, and others. It should be given in large and increasing doses, beginning in young children with a minim three times a day, and increasing to five or even ten minims. It may be given in *perles*, or in pills or in mixture; in the latter a convenient way is with tincture of gentian, alcohol, and sherry. As a rule, it is well borne by the mouth. It may also be given in the form of inhalations, the so-called *vapor creasoti* consisting of creasote, 80 minims, light carbonate of magnesium, 30 grains, water to one ounce; a teaspoonful in a pint of water at 140°. Inhalations with this are strongly recommended. Intrapulmonary or intratracheal injections of creasote in oil have been practised. The active principle of it, guaiacol, has been much used, both by the mouth and hypodermatically. Given in solution, it may be made up with tincture of gentian, rectified spirits, and sherry. Hypodermatically, it is used with sterilized olive oil, 5 per cent. solution; 1 or 2 per cent. iodoform may be employed with it, and 1 cc. of the mixture injected, gradually increasing to 3 cc. or even 4 cc. One rarely sees bad effects from creasote: the beneficial results are most marked in individuals who can take large quantities and who can enjoy the associated action of fresh air and a good diet. Creasote without these accessories is not of very great service, as witnessed in ordinary hospital practice. Patients are remarkably tolerant of it, and one rarely sees any ill effects. Other balsamic substances, such as eucalyptol, terebene, terebinthine, thymol, and menthol, have been recommended.

Symptomatic Treatment.—In this we shall refer more particularly to pulmonary tuberculosis.

The fever of tuberculosis is serious and obstinate. It will be found in the early stages that the combination of rest with fresh air is the most beneficial.

The child may be wrapped up and taken into the fresh air for the greater part of the day. We have no thoroughly satisfactory medicinal means for reducing the temperature. Antipyrine, antifebrin, and acetanilide, if used at all, must be given with great care. Quinine and salicylic acid are still used by many practitioners. When the temperature is persistently high in the early stages of tuberculous broncho-pneumonia, cold in various forms will probably be the most efficient measure, and by careful sponging the temperature may be reduced several degrees. The most satisfactory antipyretic is found in the fresh air, more particularly the change to a resort such as the Adirondacks or Colorado.

In the chronic pulmonary tuberculosis of children, when the fever is of a hectic type, sweating is a very troublesome and disagreeable symptom, for which atropine, aromatic sulphuric acid, and tincture of *nux vomica* may be used. In young children great care should be taken to prevent the chilling of the body after a profuse night-sweat. For the cough, if troublesome at night, paregoric or small doses of Dover's powder may be used. Codeine or, in extreme cases, small doses of morphine may be given. Where there is marked tenderness on the chest or pleuritic complications the cough is sometimes relieved by mild counter-irritation or the application of a warm poultice. Inhalation of terebene and oil of eucalyptus may sometimes diminish the profuse expectoration.

Hæmoptysis in the pulmonary tuberculosis of young children is usually a terminal and fatal symptom, quickly beyond treatment.

The diarrhœa may demand very careful regulation of the diet, and if profuse the acetate of lead, alone or with opium, may be used. Preparations of tannin and gallic acid are also beneficial. In all tuberculous processes there is a more or less marked tendency to anæmia, and many patients improve quickly under the administration of iron. Careful attention should be paid to the gastric symptoms. If the digestion is poor, dilute hydrochloric acid may be used, and if heartburn and pain be present some time after eating, the carbonate of sodium or the alkaline mineral waters.

MALARIAL FEVER.

BY W. S. THAYER, M. D.,

BALTIMORE.

Synonyms.—Intermittent fever; Swamp or Marsh fever; Paludism or Paludal fever; Fever and ague; Chills and fever.

The term "malaria," which has been applied in a general way to a variety of febrile and non-febrile processes, must now be limited to a certain definite class of febrile affections which we know to have a specific infectious origin. The specific micro-organisms which are the cause of these processes belong to the class of protozoa and inhabit the blood of the infected individual.

Etiology and Pathology.—The *geographical distribution* of the malarial fevers is a point of considerable interest, particularly inasmuch as it is not entirely constant. In Europe, France, Germany, and England are comparatively free from malarial fever, while in Southern Russia and Italy the disease is very frequent. In many parts of Africa and India some of the severest forms of malaria are seen. In this country there are various localities in which malaria is endemic, particularly in certain regions in the Southern States, in Louisiana, Mississippi, Arkansas, and Texas. In the low, marshy lands along the coast throughout the Southern and Central States there are many places in which malarial fevers are common. In parts of New England malaria also occurs, particularly in the Connecticut Valley, while of late a considerable number of cases has been seen along the course of the Charles River in Massachusetts. In New York City the disease is rare, though certain low-lying districts in the neighborhood give rise to a number of cases. In Philadelphia the disease is perhaps more frequently seen, but most of the cases in that city come from outlying districts. In parts of Baltimore also malarial fever occurs, though a great majority of the cases come from the districts bordering on Chesapeake Bay. In the Western States malaria is less common, but in certain parts about the Great Lakes it is more or less prevalent.

A very interesting point in connection with the geographical distribution of malarial fever is the manner in which the disease wanders from one region to another, diminishing greatly in intensity or almost dying out in a district where it has formerly been endemic, and developing perhaps in a region where it has been for many years an unknown disease. An instance of this is the appearance during the last five or six years of malarial fever along the basin of the Charles River in Massachusetts, where it had been for many years unknown. Again, in districts in which malarial fever has for years been endemic there seem to be cycles in which the intensity of the process increases and diminishes.

Malarial fever is particularly prevalent in low, swampy, and badly-drained districts, and especially in areas which are rich in vegetable matter and have

been allowed to fall out of cultivation. It is much more prevalent in tropical or semitropical regions, and is more severe in climates where the moisture is considerable. It has been thought that winds have possibly some connection with the carrying of the contagion; for instance, in some malarial districts the residents on one side of a stream may be relatively free from the disease, while those upon the other side, toward which the prevailing winds blow, may suffer considerably. The danger of contracting malarial fever is apparently greater among those living in the lower stories of a house than in the upper.

In temperate climates the frequency of the malarial fevers varies greatly with the *seasons*. The majority of cases occurs in the late summer and fall, though a certain number develops in the spring and early summer, while in the winter it is very rare. In tropical climates, where the disease occurs all the year round, the greater number of cases is seen in the fall and spring months.

THE SPECIFIC MICRO-ORGANISM.—All our accurate knowledge of the causal element of malarial fever dates from the discoveries of Laveran in 1880. While studying malarial fever in Algiers, Laveran discovered certain pigmented bodies in the blood of affected individuals. These bodies had long been observed by others, and by some accurately described, and even pictured, but, while the older observers considered them to be altered blood-corpuscles, Laveran recognized them as parasites, and asserted that they were the definite exciting agent of malarial fever. These discoveries have been confirmed by numerous other observers in Italy, the United States, Russia, Germany, and India. In this country Councilman, Abbott, Osler, James, and Dock have made valuable observations. Laveran and his school have published careful and accurate descriptions of the different forms of the parasite, which may be seen in the blood, but they assert that they are unable to associate any definite types of organism with distinct types of fever. From the observations which have been made, however, by the numerous Italian observers, led by Golgi, there can be to-day little doubt that certain definite types of the organism are associated with certain definite types of fever.

In this country, as in Italy, there are several main types of fever:

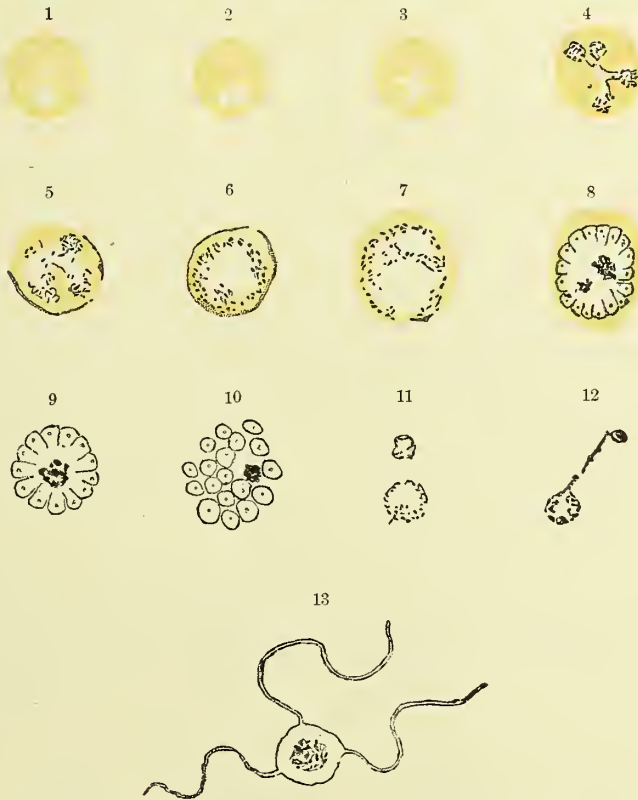
(1) The milder forms of intermittent fever, which form the great majority of the cases in the spring and early summer, but which occur at all malarial seasons: (*a*) tertian and double tertian (quotidian) fever; (*b*) quartan fever, with its combinations.

(2) The more severe, often more or less irregular, fevers which occur here, as in Italy, more commonly in the later summer and fall—the *æstivo-autumnal* fevers of the Italians, the tropical malaria of the Germans. This type of fever includes the so-called remittent malarial fevers as well as most of the cases of pernicious malaria and of the malarial cachexiæ. Some of the Italian observers have attempted to divide these fevers, again, into (*c*) quotidian fever, and (*d*) malignant tertian fever. In this country, however, we see probably only the quotidian type. With each of these types of fever is associated a distinct type of the specific micro-organism.

(*a*) *The Parasite of Tertian Fever.*—Golgi was the first observer who accurately described and differentiated the organisms of the tertian and of the quartan forms of malarial fever, and his admirable observations have remained practically unassailed. If we examine the blood from a case of tertian fever just after the paroxysm, we find in certain of the red blood-corpuscles small round, colorless bodies (Fig. 1, ¹, ², ³) which appear to have a slight depression in the centre, and when stained in dried specimens show a paler central area with a darker periphery. These bodies, examined in the fresh specimen,

show active amoeboid movements. A few hours later the organism will be found to have increased somewhat in size, and to contain a few fine brownish pigment-granules which dance actively under the eye (Fig. 1, 4), the motion probably being due to undulatory movements in the protoplasm. On the day between the paroxysms the bodies will be found to have about half filled the red corpuscle (Fig. 1, 5). They are still actively amœboid, and the number of pigment-granules has considerably increased. The red corpuscle at this stage will be seen to be a trifle larger than its unaffected neighbors, and to be considerably decolorized. On the day of the paroxysm

FIG. 1.



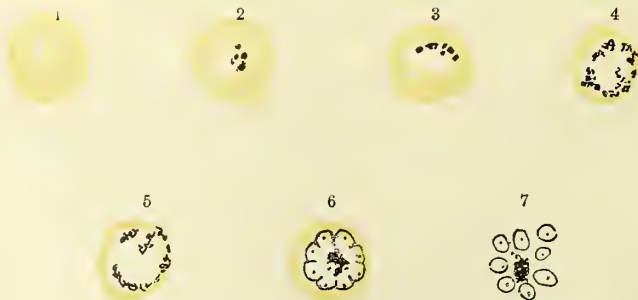
The Parasite of Tertian Intermittent Fever (drawings made from the blood of patients in the Johns Hopkins Hospital, with the camera lucida. Winckel, 1-14 oil immers. lens, 4 eye-piece): 1, 2, 3, hyaline intracellular amoeboid bodies, seen during the febrile stage of the paroxysm; 4, 5, half-grown bodies seen on the day between paroxysms; 6, the same, further advanced; 7, full-grown body seen during the paroxysm; 8, segmenting body seen during the paroxysm; traces of the red corpuscle still seen about the organism; 9, 10, segmenting border further advanced; 11, 12, extracellular pigmented bodies, regenerative forms; 13, flagellate body (somewhat diagrammatic, not drawn with the camera lucida).

the organism has entirely filled and almost destroyed the red blood-corpuscle, which is represented only by a faint pale rim about the full-grown parasite, if indeed it has not entirely disappeared (Fig. 1, 7). The pigment-granules may show at this stage a very active motion, but the amoeboid movements of the organism as a whole are but little marked. At the time of the paroxysm an interesting change takes place; the pigment gathers together in a more or less solid clump, usually in the centre of the organism, while the rest of the protoplasm looks somewhat granular and shows a suggestion of

lines radiating outward from the centre (Fig. 1, ⁸). This appearance gradually changes, the lines becoming more distinct (Fig. 1, ⁹), until finally we see the central clump of pigment surrounded by from fifteen to twenty small ovoid or round glistening segments, each one having a central more refractive spot, and resembling strongly the hyaline bodies which we see immediately following the chill (Fig. 1, ¹⁰). This segmentation of the organism is always coincident with the paroxysm, and the presence in the blood of a segmenting body is a sure indication that the paroxysm is present, or is about to occur. Immediately following the paroxysm fresh hyaline bodies appear in the red corpuscles. Though the invasion of the corpuscles by these fresh segments has never been actually observed, the evidence that this occurs is so strong that we can safely accept it as a fact. Besides these forms we see not infrequently small or large extra-cellular pigmented bodies; that is, organisms resembling exactly those within the red blood-corpuscles, excepting that they are free in the blood-current (Fig. 1, ^{11, 12}). These may be seen at times to break up into several smaller bodies, while at other times they may show a long, tail-like, non-motile process, containing sometimes a few pigment-granules. They are probably organisms which have escaped from the red corpuscles, or full-grown bodies which have broken up; they are considered to be degenerative forms. At times also we find the so-called flagellate bodies. Their development from the pigmented organism may indeed be observed, the pigment of the full-grown body becoming very actively motile, then collecting in the centre of the organism, while several long, thread-like flagella burst out of the body and move actively about among the surrounding corpuscles (Fig. 1, ¹³). Sometimes we may see one of these flagella which has broken away from the organism and is moving rapidly through the field. This is also thought by the Italians to be a degenerative process. The characteristics of this form of organism, which is observed in tertian fever alone, are so marked that with a little study of the parasite one can make a definite diagnosis of the type of fever from an examination of the blood alone.

(b) *The Parasite of Quartan Fever.*—Quartan fever is not at all common in this country, but in the few cases which the writer has observed the organisms differ distinctly from the tertian parasite, and show accurately the characteristics described by Golgi. Here the first stage of the organism is similar to that observed in tertian fever, excepting that the amœboid move-

FIG. 2.



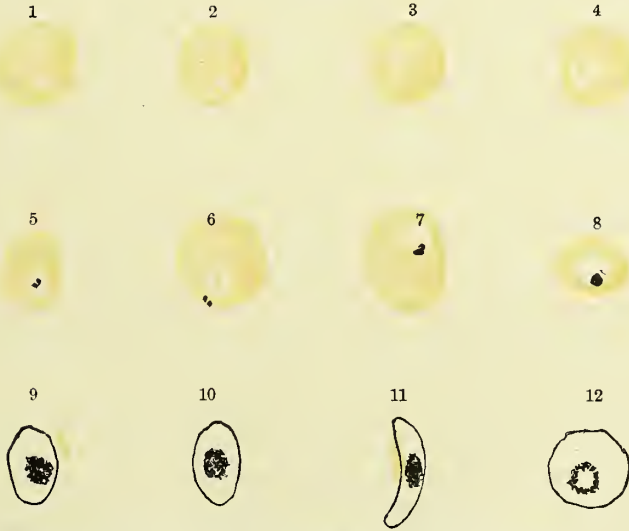
The Parasite of Quartan fever (drawings mainly after Marchiafava, Bignami, and Mannaberg): 1, hyaline amœboid intracellular body; 2, 3, 4, further stages in the growth of the body; 5, full-grown form; 6, 7, segmenting bodies.

ments are not so active. As the body develops the rods and clumps of pigment are larger and darker than those in tertian fever, while the amœboid

movement of the organism is relatively slight. The full-grown forms are materially smaller than in tertian fever, while the red blood-corpuscle, instead of being expanded and decolorized, appears at times shrunken about the body, and of a somewhat deeper old-brass color (Messingfarbe). In segmentation the organism divides into from six to ten different parts instead of twenty or thirty, as in the tertian form (Fig. 2, 1-7).

(c) *The Organisms of the Æstivo-autumnal Fevers.*—The organisms associated with the æstivo-autumnal fevers have been carefully studied, but much remains to be done, particularly in this country. There is some difference of opinion as to whether there are not two types of organism associated with these fevers. Some Italian observers divide them into the quotidian and the malignant tertian organisms. The differences made out by the Italians are, however, very slight, and have not been observed in this country. In the first place, we see just after the paroxysm small hyaline bodies which may or may not be actively amœboid; these can sometimes be distinguished from those appearing in the initial stage of either tertian or quartan fever, in that they are generally somewhat smaller and have oftentimes a characteristic ring-like appearance (Fig. 3, 1-4). In the early stages—during the first week, for instance—of an attack of this form of fever we may see only the hyaline, unpigmented forms, but commonly, if we observe carefully, we may see, some time after the exacerbation of temperature, shortly before the beginning of another, bodies which are a trifle larger than these smallest hyaline forms, and which contain one or two very minute pigment-granules lying near the periphery (Fig. 3, 3, 4). Just before or during the paroxysm we may see

FIG. 3.



Parasites seen in Æstivo-autumnal Fever—tropical malaria. (Drawn with the camera lucida from the blood of patients in the Johns Hopkins Hospital; Winckel, 1-14 oil immersion lens, 4 eye-piece.) : 1, 2, 3, hyaline, ring-like amœboid bodies seen in the blood toward the end of the paroxysm; 4, the same further developed; 5, 6, disc- and ring-shaped bodies with one or two small pigment-granules, seen shortly before a paroxysm; 7, full-grown body with central pigment-granules, seen during paroxysm; 8, full-grown body with central active pigment-corpuscle crumpled and shrunken; 9-12, crescentic and ovoid bodies with coarse central pigment. 9 and 11 show remains of the corpuscle (from a case of chronic malaria with normal temperature).

bodies with a small central clump of motile or non-motile pigment-granules lying usually in cells which are more or less shrunken and crumpled, and of a deeper color than the normal corpuscles (Messingfarbe). These bodies are

generally not half as large as the red corpuscle (Fig. 3, ^{8, 9}). After the first week or ten days of the disease, or after treatment has been begun, we see, however, certain very characteristic and easily recognizable forms which are only seen with this type of fever. These are, first, round or ovoid bodies about the size of a red blood-corpuscle, a little smaller or a little larger, with clear, rather highly refractive, waxy-looking protoplasm, and coarse dark pigment-granules, which are usually collected in a ring or a mass in the centre of the organism (Fig. 3, ^{9, 10, 12}). The granules are usually very slightly motile. At one side of the body we often see a small bib-like attachment which may show a slightly yellowish color. On examination this proves to be the remains of the red blood-corpuscle in which the organism has developed. In association with these are seen crescentic bodies (Fig. 3, ¹¹), the protoplasm of which shows the same characteristics as that in the forms above described, while the pigment is collected in the middle in a similar ring or bunch, and is but slightly motile. On the concave side of these crescents one may also often see a bib-like attachment, just as in the ovoid forms. At times during the examination of the fresh specimen we may see the change from an ovoid body into a crescent take place. The development of these forms from the hyaline bodies can be followed out on careful observation. They are thought by some to be a resting stage of the organism. Segmenting bodies are almost never seen in the circulating blood of this form of malarial fever, though the presence of the round intracellular bodies with central pigment is a sure sign that segmentation is going on elsewhere. It has been found by the Italians that after the accumulation of a few pigment-granules the organisms seek the internal organs, where segmentation takes place. The bodies are still small and contained within the red corpuscle. The pigment gathers in the centre, as in the other types of segmentation, while the segments are very small and rarely more than twelve in number. During the paroxysm we may see large numbers of leucocytes containing pigment granules and clumps which are probably the remains of segmenting organisms. Flagellate bodies may be observed here as in the tertian and quartan fevers, but only when ovoid and crescentic pigmented bodies are present. They may be seen to develop from the round bodies with central pigment.

Careful studies concerning the morphological characteristics of the malarial parasite have shown that it belongs to the class of Protozoa, and is possessed of a nucleus containing one or more nucleoli. At the time of sporulation this nucleus divides—according to some directly, according to others by karyokinesis.

Pathological Anatomy.—In the acutely fatal cases of malarial fever (pernicious malaria) certain fairly characteristic changes are found in the various organs.

The *brain* may show few changes. At times, however, there may be a slight subpial cedema, with hyperæmia of the cerebral substance and perhaps punctate hæmorrhages. Melanosis may be entirely absent. Microscopically, however, the changes are most characteristic. The cerebral capillaries are crowded with malarial parasites, which may be in all stages of development, though generally one of these phases is most marked. At times the organisms may not be so numerous, but free clumps of pigment may be found, and large endothelial cells and leucocytes containing pigment-clumps and red corpuscles. There is usually a marked granular and fatty degeneration of the endothelium of the vessels, a change upon which the punctate hæmorrhages may depend. These lesions are particularly marked in the comatose forms of pernicious malaria. In other forms the cerebral lesions may be much less marked.

The *spleen* is always enlarged: the capsule is tense; the parenchyma is cyanotic, of a slaty-gray color, and almost diffuent. In some cases of acute malaria death may occur from rupture of a greatly enlarged spleen. The pulp contains enormous numbers of red blood-corpuscles, many of which contain parasites. It also contains numerous large white elements rich in protoplasm, containing usually a single bladder-like nucleus, and at times coarse granulations. These elements are usually laden with pigment, which at times has the same arrangement as it does in the body of the parasite itself. Sometimes these cells may contain the entire red corpuscle with the organism. There may be free pigment in the intercellular spaces of the pulp. The small mononuclear elements and the lymphocytes of the follicles never contain pigment. The capillaries are usually filled with the plasmodia, while the splenic veins show relatively few, though they always contain large cells enclosing pigment or the remains of red blood-corpuscles.

The *liver* has usually a slaty-gray color. There is always cloudy swelling, while microscopically small areas of necrosis have been described by Guarneri. The capillaries are filled with leucocytes which contain numerous pigmented bodies. Relatively few plasmodia are found in the blood-corpuscles in the vessels. Numerous liver-cells are found containing clumps of hæmatin and altered red corpuscles—a condition similar to that which has been found in pernicious anæmia, which, as Bignami suggests, may explain the polycholia which is commonly found in subjects who have died of pernicious malaria. On this probably depends the icteroid hue in severe malaria.

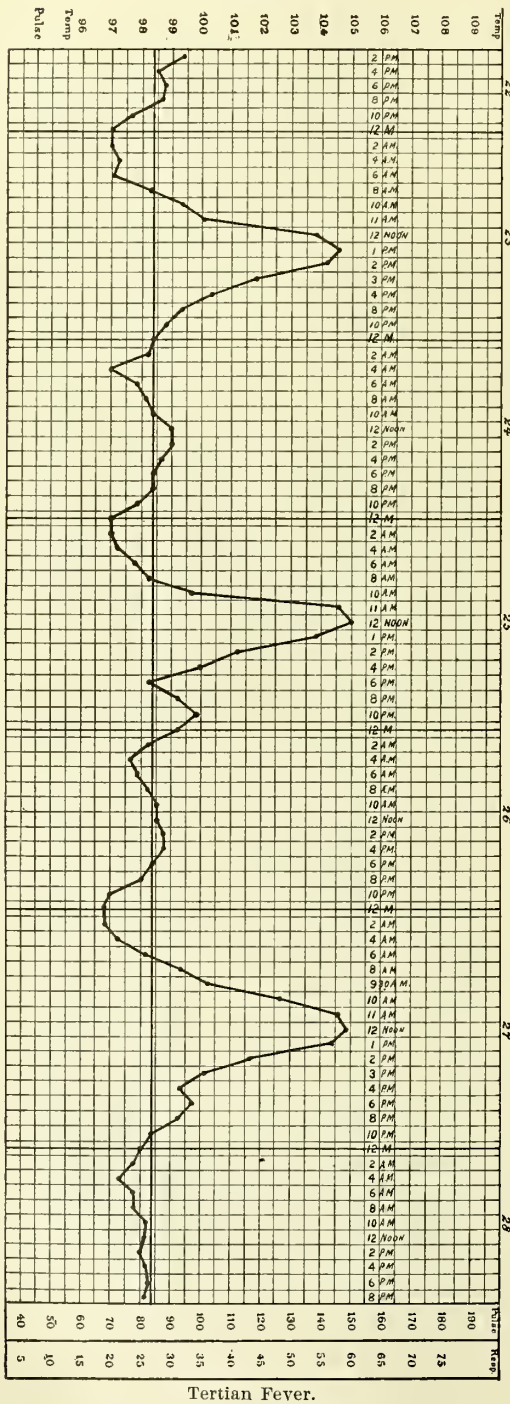
The *lungs* show in their capillaries numerous cells containing pigment-clumps and well-preserved parasites, though it is unusual to find pigment in the endothelial cells, in the capillaries, and smaller veins. In the areas of broncho-pneumonia which may occur, polynuclear leucocytes are chiefly found, while the large pigmented cells take no part apparently in the active inflammatory process.

The vessels of the *kidneys* contain relatively few organisms. The glomeruli may be considerably pigmented. There may be marked degeneration of the epithelium of the capsules, and at times changes in the parenchyma, especially areas of necrosis of the epithelium of the convoluted tubules. The other viscera show no especially characteristic changes excepting at times the melanosis.

In the more *chronic forms of malaria* and in *malarial cachexia* the anæmia is usually particularly marked. The *spleen* is always enlarged and very firm. There is a marked thickening of the capsule, which is often adherent to the neighboring tissue. On section the spleen is generally of a dark brownish-gray color, the fibrous tissue throughout the organ being greatly thickened. The *liver* is considerably enlarged, and usually has a grayish-brown or slaty color. Microscopically, Kupfer's cells and the perivascular tissue may contain much pigment. At times there is a considerable increase in the connective tissue. The *kidneys* show no particular characteristic changes, though there may be considerable pigmentation; the pigment is most marked about the blood-vessels and the Malpighian bodies, and sometimes in the region of the convoluted tubules. There are no characteristic changes in the other organs, excepting the slaty-grayish pigmentation.

Symptoms.—As may be gleaned from what has already been said concerning the specific organisms, malarial fever occurs in several main types: (1) The milder intermittent fevers, which form the majority of all cases in the more temperate climates, and occur in the warmer climates more commonly in the spring and early summer: (a) Tertian intermittent fever and its combinations; (b) Quartan intermittent fever and its combinations. (2) The more irregular, æstivo-autumnal fevers, which usually show quotidian paroxysms.

FIG. 4.



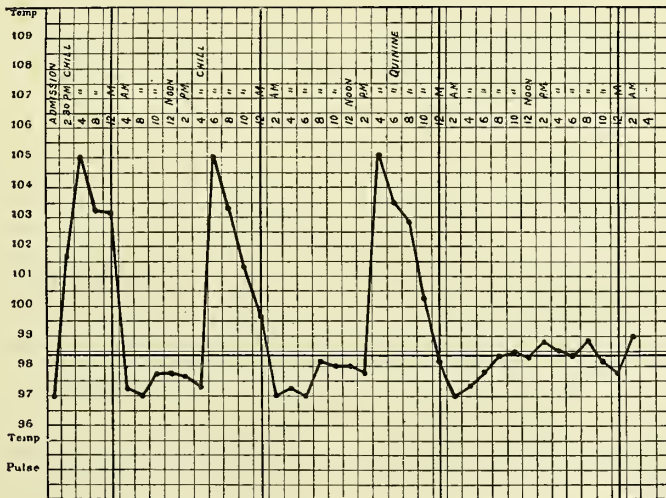
TERTIAN INTERMITTENT FEVER.—This is by far the commonest form of malarial fever in this country, and with the quartan fever forms the mildest type of the disease. It is the type of the intermittent fever of the spring and early summer, though it may be seen at any time of year. It shows often no particular tendency to increase in severity, while in many instances, under proper care and change of climate, spontaneous recovery may occur. It depends, as we have seen, upon the invasion of the blood by an organism which passes through its cycle of existence in forty-eight hours. The febrile paroxysms occur when these parasites have reached their full development and begin segmentation. These periods occur with considerable regularity at intervals of forty-eight hours one from another. In older children the paroxysms may usually be divided into three stages: first, the *chill*; secondly, the *fever*; and thirdly, the *sweating*. The child, who may have been feeling fairly well beforehand, becomes suddenly uneasy, may begin to yawn, or may have an attack of vomiting or diarrhoea, which is followed or accompanied by a well-marked rigor, associated with cyanosis and coldness of the extremities. The temperature rises to a considerable height, possibly to 108° F. This stage lasts for a varying time, from ten minutes to an hour. As the chill ceases the patient passes into a stage in which there is marked flushing of the skin, with great heat and dryness. The child complains bitterly of thirst and

headache, and is usually very fretful. There may be, as in the first stage, renewed attacks of vomiting or diarrhoea. This stage, after lasting for a vari-

able length of time, from half an hour to three or four hours, is followed by profuse sweating, the temperature falling within an hour or two to a normal or even a subnormal point. With the sweating the child may seem exhausted and weak, but shortly afterward appears again perfectly well.

Such an attack as this differs but little from the intermittent fever of adults, and indeed above the age of six the differences are very slight. Under this age, however, there are marked differences in the paroxysm. Very commonly in young children both the first and the third stages, those of the chill and sweating, are absent. The first stage is then generally represented by a slight restlessness, the face looks pinched, the eyes sunken, the finger-tips and toes may become cyanotic and cold, while the child may yawn or stretch itself. Oftentimes there is nausea or vomiting, and possibly diarrhoea. This may be the only manifestation of the first stage, though it may be followed by slight or severe nervous symptoms. These begin usually with a slight spasmodic twitching of the eyelids or of the extremities, and may go on to general convulsions. The chill in the adult is very often represented in the young child by the convulsion—a fact which is as true in all other acute febrile processes as in malarial fever. This stage lasts usually for a short time, not more than an hour or so. The temperature rises rapidly, possibly to 108° F.; then comes the period of fever, during which the child is much flushed, is restless, thirsty and fretful, while, as has been already said, various gastro-intestinal disturbances may occur. The fever remains at its height for an hour or two; afterward there is a gradual fall of temperature, unaccompanied by sweating. In many instances, besides the slight coldness of the hands and blueness

FIG. 5.



Double Tertian (quotidian fever).

of the finger-tips, and a somewhat pinched expression of the face in the first stage, the first and the third stages of the attack may be entirely lacking.

Pure tertian fever is rare in children, as the process is almost always a double infection; that is, the blood contains two sets of organisms, which attain maturity on alternate days, and give rise to *quotidian* paroxysms. If,

as is unusual, the case is one of pure tertian fever, the child may seem perfectly well on the day between the attacks.

Physical examination during the very first attack may reveal little or nothing, but usually by that time, and always after one or two paroxysms, an enlarged spleen may be made out. If a child has had more than two supposed malarial paroxysms and the spleen is not distinctly enlarged, we have almost sufficient evidence to put aside the diagnosis of malarial fever. Herpes labialis is a very common accompaniment. Anæmia is usually noticeable if the process has lasted for any length of time. The discovery of the specific organism in the blood is the one diagnostic point. The paroxysm in tertian malarial fever may last altogether from twelve to fifteen hours, though commonly it is much shorter, the first stage lasting from ten minutes to an hour, the second stage from an hour to three or four hours, and the third stage a varying length of time. As the length of time which the tertian organism requires to attain its full growth is almost exactly forty-eight hours, the attacks dependent upon one group of parasites occur almost regularly forty-eight hours apart, though in some instances we may find a tendency to anticipation or to retardation in the attacks. This point can only be determined by observation, so that one cannot definitely prophesy the hour at which an attack will occur until he has seen several paroxysms. It is easy to see that in the quotidian cases, which depend upon the presence of a double infection, the chills on the alternate days may occur at different hours, one group of organisms segmenting perhaps at ten o'clock, and the other at two. Usually, however, these differences are slight. Not infrequently we find the history of tertian attacks at first, and later on daily attacks of fever. The commonest time for the paroxysm in tertian fever is in the early part of the day, between eight in the morning and two in the afternoon, though they may occur at all hours either of the day or night. Irregularities in the course of the fever, no matter what the type may be, are much commoner in children than in adults.

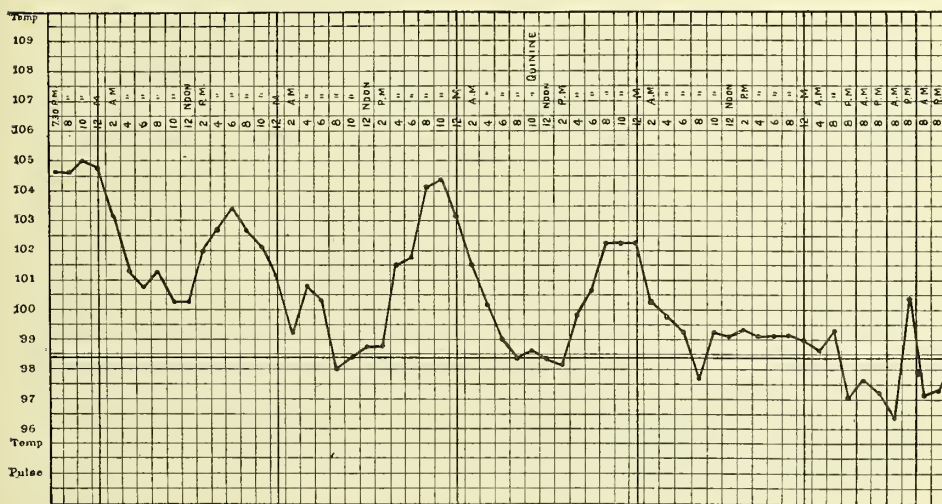
QUARTAN FEVER.—This form of fever is rarely observed in this country. Out of about 500 cases of malaria treated at the Johns Hopkins Hospital in four years, it only occurred twice. Here the length of time required for the development of the organism is seventy-two hours, and the paroxysms occur every fourth day. The nature of the paroxysm does not differ from that observed in tertian fever. As one may easily see, complex attacks of fever may arise from a double or triple infection with quartan organisms. Thus we may have a daily paroxysm due to a quartan infection, or, on the other hand, paroxysms on two days in succession, with one day intermission, a triple or a double infection. The diagnosis of quartan fever may be made by a skilled observer from one examination of the blood by the discovery of the characteristic quartan organism.

THE ÆSTIVO-AUTUMNAL FEVERS. "**TROPICAL MALARIA.**" "**FEBRIS IRREGULARIS.**"—The malaria occurring in the late summer and fall is often of a much more severe type than that occurring in the spring, and, as has been shown by the Italian observers, most of these cases are due to a different type of the specific organism. It is in the later summer and fall that we see most of the cases of apparently irregular fever, and the so-called remittent malarial fever. The typical malarial cachexia, while it may follow any form of intermittent fever, usually results from this type of malaria. Most of the pernicious forms also come under this heading.

THE SO-CALLED IRREGULAR REMITTENT FEVERS.—The recent Italian observers, asserting that there is in reality no actual irregularity, divide these

fevers into the quotidian, in which a daily paroxysm occurs, and the tertian, in which the paroxysm occurs on every other day; but in both instances there is a greater tendency to irregularity in the time required for the development of each brood of organisms. On the one hand, there is often a very marked tendency for the paroxysms to anticipate one another, or there may be a retardation, while again the attacks do not present themselves in so clean-cut and regular a form as in the spring fevers. They may be much lengthened out, so that one attack may follow another without the temperature ever actually reaching a normal point. Most of the cases of this type of fever seen

FIG. 6.



Æstivo-autumnal fever. (Quotidian.)

in this country show a distinct daily paroxysm; it is doubtful whether we see in America the "malignant tertian fever" of the Italians. The attacks may differ little from those in the ordinary tertian form, excepting that they are often more severe and of a somewhat longer duration, so that the afebrile periods are shorter or even absent. On the other hand, the onset may be very gradual, with daily exacerbations of temperature, accompanied by restlessness, flushing, often vomiting or diarrhœa, and headache, but without chills or perhaps even sweating. The attacks may be prolonged and run into one another, so that a remittent temperature results. There is often delirium or drowsiness and somnolence; the spleen is always enlarged. In this condition the diagnosis from typhoid fever or meningitis may be impossible without an examination of the blood. Such cases as this, however, do not generally go on to recovery without treatment, but tend to become pernicious, the paroxysms increasing in severity till death.

MALARIAL CACHEXIA.—The fever in some instances may never rise as high as it does in the paroxysms of tertian fever, nor may the immediate symptoms of the paroxysm be as striking, and the attention of the physician is often called to the patient for the first time when the stage of malarial cachexia has been reached. The child may then show a pitiful appearance. It is pale, of a sallow, parchment-like color, and often much emaciated. The skin is dry, the face has a drawn, pinched look, the eyes are sunken; there may be marked symptoms on the part of the digestive tract, frequent attacks of vomiting and

diarrhœa. The fever may stand in the background. Indeed, in some of these cases there may be for weeks relatively little fever. The spleen is always enlarged. Malarial cachexia does not exist in children without an enlarged spleen. In all instances, no matter whether our attention is called to the child on account of the fever or of the gastro-intestinal derangement, an examination of the blood will show the organisms, usually those characteristic of the æstivo-autumnal or tropical malarial fever, the small hyaline bodies, and the pigmented crescents and ovoid forms. Malarial cachexia may follow all forms of the disease, and not infrequently is seen in improperly treated cases of tertian fever or in those who have been subject to repeated attacks, but it is much more commonly seen in this type of fever.

PERNICIOUS MALARIAL FEVER.—It is in the æstivo-autumnal fevers that we see more commonly the pernicious forms of malaria, though these are rare in temperate climates. In these cases a previously healthy child may begin to show a slight restlessness, with a pinched expression of the face and some blueness of the extremities. An attack of vomiting or diarrhœa may occur, which may be followed suddenly by severe convulsions and a very rapid rise in temperature, which may be as high as 108°. The convulsions may continue or the child may pass into a dull, comatose condition, the pupils being fixed and possibly irregular; in this condition it may remain until death ensues. In some instances the whole attack may be represented by a condition of coma with collapse, possibly with little or no rise in temperature. These severe attacks are rare in this country, and it is not at all improbable that in regions in which severe malarial fever prevails many non-malarial attacks are ascribed to this disease. The definite diagnosis can only be made by the discovery of the parasite in the blood. Some of the most severe of these attacks are probably due to the infection with several groups of the organisms at once, so that segmentation is going on continuously.

AFFECTIONS OF OTHER VISCERA SOMETIMES ASSOCIATED WITH MALARIAL FEVER.—*Respiratory Apparatus.*—In all forms of malarial fever bronchitis is a common complication, as it is, indeed, with any acute febrile affection. This is particularly true in children. The appearance of a profuse coryza in the absence of the sweating stage has been noted.

Alimentary Tract.—In almost all cases of malarial fever in children symptoms are present on the part of the stomach and intestines. Vomiting in the first and second stages of the paroxysm is extremely common, while diarrhœas are also very frequently seen in all forms of malaria, particularly in the more remittent forms and in the chronic cachexia, where it is probably generally due to a secondary infection to which the debilitated child is more readily subject. Little is to be noticed on the part of the circulation.

Kidneys.—Slight albuminuria may often be observed, and in rare instances hæmaturia occurs. Malarial hæmaturia is generally considered a grave symptom. It is probably, however, a rare condition, except in districts where the severest forms of the disease are common. Many of the so-called malarial hæmaturias are due to other causes.

The literature of malarial fever contains numerous references to "malarial pneumonia," "malarial bronchitis," "malarial neuralgia," "malarial diarrhœas," and the like, most of which, in the light of our present knowledge, have probably little or no connection with malarial fever. It is easy to understand that the child debilitated by a severe malarial fever may more readily fall a victim to a variety of other diseases. In this way probably the gastro-intestinal and bronchial disturbances so commonly observed are to be explained. That there is any such thing, for instance, as a specific malarial

pneumonia is wholly out of the question. The chills which may occur sometimes with some regularity in the course of many of the specific fevers are commonly attributed to a malarious influence. These inferences are for the most part unjustifiable. In rare instances a patient who is subject to an acute or chronic malaria may develop typhoid fever at the same time, or the converse may occur, but these instances are few and far between, and the great majority of instances of chills occurring in typhoid fever have no connection whatever with malaria. Pneumonia may develop during the course of a malarial attack, but it is due in these cases to its specific cause. The examination of the blood is our one safe clue to the explanation of such complications.

Diagnosis.—*The Milder Tertian and Quotidian (double tertian) Fevers.*—The diagnosis of malarial fever in children may be made, in the first place, from the character and periodicity of the attacks; secondly, from the enlargement of the spleen, which is always present after the first or second attacks; and thirdly, by the presence of the malarial organism in the blood. In some instances there may be relatively few parasites, but the careful examination of several fresh specimens of the blood will always reveal the organism if present. Even in the absence of definite data with regard to the attacks, the diagnosis may be made by the type of organism found. The commonest type, as has been said, is the double tertian, *quotidian* fever.

The commonest condition with which malarial fever is confounded is tuberculosis in its various forms; the hectic evening temperature is often ascribed to malaria. Most pediatricists may, I fancy, remember more than one instance where after a diagnosis of malarial fever evidences of pulmonary, abdominal, or even glandular tuberculosis have developed. The absence of definite signs of tuberculosis, the splenic enlargement, and the anæmia, which may be marked, speak in favor of the malarial nature of the affection, while the absence of malarial organisms in several specimens of fresh blood, even in the presence of marked febrile paroxysms, is a sure sign of the absence of malarial fever.

The same rules of diagnosis apply to *quartan* fever. The characteristic organism of that type will be found on examining the blood.

Æstivo-autumnal Fevers.—It is the more irregular and remittent fevers and the malarial cachexiæ which give the most trouble to the diagnostician. The regularly intermittent fever may not here give us our clue to the diagnosis. On the other hand, the presence of a considerable anæmia in association with a markedly enlarged spleen, which is always present in this form of fever, will lead us to suspect the proper diagnosis, which will be confirmed by the discovery of the small ring-like hyaline intracellular organisms, and, if the case has lasted a week or more, the ovoid and crescentic pigmented bodies in the blood. This form of fever may often be confounded with tuberculosis. It may also simulate very closely, from the physical examination alone, leukæmia or the anæmia infantilis pseudo-leukæmica of Von Jaksch. In some instances where the paroxysms tend to run into one another and produce a more or less remittent fever, the differentiation of the process from fever may be impossible from the physical examination alone. The frequent herpes, the large size and prominence of the spleen, as well as the rapidly developing anæmia, may be suggestive, but here, as elsewhere, the examination of the blood alone gives us our certain diagnosis. In the absence of an examination of the blood, the chronic cachexiæ may be considered to be the result of the concomitant gastro-intestinal derangements or of the bronchitis, while in many instances the atrophy, the dyspepsia, and the diarrhœa may be found to depend upon the presence of the malarial organisms in the blood. In the cases of

severe pernicious malarial fever the examination of the blood is also our only safe clue to a diagnosis.

METHODS OF EXAMINATION OF THE BLOOD.—The examination is best made with fresh specimens. The lobe of the ear is punctured with a sharp, spear-pointed lancet; a very small cut is all that is necessary. This may be done behind the back without the child seeing the instrument, so that it may not be alarmed, while if the instrument is sharp the process is almost painless. In some instances it may be done while the child is asleep, without even awakening it. After wiping away the first drop or two of blood, a perfectly clean cover-glass is brought into contact with the tip of a small drop of blood, and allowed to fall immediately upon a freshly-cleaned slide. If the slide and cover-glass have been washed in alcohol just before using and are perfectly clean, the drop of blood will spread out regularly under the glass, and the corpuscles may be seen lying side by side free from crenation or any other artificial changes. Pressure on the cover-glass may spoil the specimen. It is best to hold the cover-glass in a forceps in order to avoid any injury to the corpuscles from the moisture of the hand. The specimen is then examined at best with a $\frac{1}{2}$ oil-immersion lens, and a 2, 3, or 4 eye-piece. A 4 eye-piece with an 8 objective, or a Zeiss E or F, will answer the purpose well, though an oil-immersion lens is clearer and better. In this manner all forms of the organism may be seen while yet alive. When it is impossible to examine the fresh specimen, dried and stained specimens may be used. A small drop of blood is taken upon one cover-glass, which is then allowed to fall upon the second glass. The drop immediately spreads out, and the two glasses are separated by being gently drawn apart. These specimens are allowed to dry in the air. They may be kept for almost any length of time before examining. There are numerous different methods for preparing and staining the specimen. As satisfactory a method as any is to place the glass in a solution of absolute alcohol and ether, equal quantities, for a half to one hour, or the specimens may be heated for from one to two hours at 100°–120° C. The specimen may then be stained in a concentrated aqueous solution of methylene blue for about a minute, washed in water, dried between filter-paper, mounted in balsam or oil, and examined. The red corpuscles remain unstained. Only the nuclei of the leucocytes, the malarial organisms, and occasional blood-platelets take up the blue coloring. In case a double stain is desired, one may make use of two solutions: Solution 1. Eosin 1 part; 70 per cent. alcohol 100 parts; Solution 2. Saturated aqueous solution of methylene blue. After preparing the specimen in absolute alcohol and ether as before, place it in Solution 1 for from fifteen seconds to half a minute, wash in water, dry between filter-paper; place it then in Solution 2, which has been diluted one-half with water, letting it stain for from one half to one minute; wash in water, and dry. By this method the red corpuscles and the eosinophilic granules in the leucocytes are stained red by the eosin, while the nuclei of the leucocytes and the malarial parasites are stained blue.

Good results may be obtained by Romanowsky's method: saturated aqueous solution of methylene blue, 1 part, 1 per cent. aqueous solution of eosin 2 parts. Do not shake or filter the mixture. Place the specimen (heated as above) in this mixture for two to three hours, and then in water for one to two hours, and dry. The parasites are stained blue. In this manner any practitioner who possesses a microscope may, without much labor, make the diagnosis of malarial fever. The examination of the fresh specimens will probably be found to be more satisfactory, and the observer who studies only stained specimens must beware of certain mistakes which one who is not familiar with the examination of

the blood may readily make, such as the confusion of the blood-plates, the hæmatoblasts of Hayem, with the malarial parasite—a mistake which certain good observers have recently made.

Course and Prognosis.—Excepting in the acute pernicious cases the prognosis in malarial fever is good, provided the case is recognized and properly treated.

If untreated the fever may take one of three courses :

- (1) Mild cases may go on to spontaneous recovery ;
- (2) The paroxysms may gradually diminish in intensity, the fever becoming less marked, while grave anæmia develops, and the patient passes into the condition of chronic cachexia ;
- (3) The paroxysms may increase in severity, assuming finally a pernicious type.

Treatment.—*Prophylaxis.*—In a malarial district certain prophylactic measures should be taken with children as well as with adults. The child should be kept in the house after sundown and should be carefully kept away from those regions in which experience has shown that malaria is present. Sleeping on the ground floor of houses in malarious districts should be avoided.

Medicinally, we possess in quinine one of the few specific drugs which are at the command of the physician. In almost all cases of malarial fever we may expect with confidence a complete recovery after the use of quinine. There is only one form of malarial fever, and that rarely seen in this country, the acute pernicious malaria, in which we cannot entirely rely upon this drug. In the milder forms of the disease, the tertian and quartan fevers and their combinations, small doses of quinine are rapidly efficacious. One or two grains of quinine (.065–.13), three times a day in children under six years of age, will be followed by the rapid disappearance of all symptoms. The best time to administer a single larger dose of quinine is immediately after a paroxysm. In the more chronic and irregular forms, which are so apt to occur in the later summer or fall, the forms in which the smaller organisms are found, much longer treatment and larger doses of quinine may be required. Ordinarily, however, doses larger than two or three grains (0.13–0.2) three times a day are not required under five or six years of age. Relatively large doses of quinine may, however, be well borne, and in cases of pernicious malaria must be administered. Ferreira states that he has given doses as large as 15 grains in infants under one year of age without noticing ill effects !

In pernicious cases the quinine must generally be administered hypodermatically. A good preparation is the muriate of quinine and urea. In ordinary cases it is probably better to give smaller doses several times a day than it is to give one large dose with the idea of “breaking up” the fever. In some children it is very difficult to administer quinine by the mouth, on account of the difficulty in disguising the taste, and because in some cases it is constantly vomited. In some cases in infants the drug is with difficulty retained. Here small doses should be given and often repeated. In these instances it may be administered by the rectum ; the dose under these circumstances should be about double that by the mouth. The administration of quinine through the skin by means of ointments is probably of little value. In cases of the more chronic æstivo-autumnal forms of malaria, associated with crescent organisms in the blood, the treatment by quinine may have to be continued for a considerable length of time. The crescents may be found in the blood for months. The fever, however, if the case is truly one of malaria, will surely yield to the treatment after a few days. Much has been written about those

forms of malaria both in children and adults which do not yield to quinine. These cases are probably not true malarial fever, as examination of the blood will show. Few cases of fever in this country do not yield within a few days to treatment by quinine. By this it is not said that relapses may not occur; they are frequent in cases where the treatment has been continued too short a time. In some of the acute forms of fever, and more particularly in the more chronic forms and in the malarial cachexia, the anæmia and various gastrointestinal disturbances may also demand our attention. In most instances, with proper attention to the diet, the gastro-intestinal symptoms will disappear after the disappearance of the fever. The anæmia, however, may require extended treatment with various preparations of iron, and even in the severe cases with arsenic, which is particularly well borne by children. The administration of arsenic, which is common in chronic malaria, has its chief value in its effect on the anæmia. Various other drugs have been tried in malarial fever, some of which have some influence on the attacks. The most important of these are preparations of eucalyptus and, of late, methylene blue. None, however, approach quinine in efficacy.

One attack of malarial fever does not, unfortunately, render the patient immune. On the other hand, he seems, if anything, to be more readily subject to fresh attacks, and in some instances these attacks may be so frequent and prolonged that a removal of the child to a proper climate is necessary.

PART IV.

GENERAL DISEASES NOT INFECTIOUS.

RACHITIS.

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NEW YORK.

RACHITIS is a constitutional disease, but its most conspicuous anatomical characters pertain to the osseous system. The gross nutritive changes which it produces in the bones and cartilages, causing deformities, are well known to physicians and the laity. In addition to these anatomical changes in the skeleton, typical cases exhibit a lack of tonicity with stretching of the ligaments, causing the knock-knee and flat-foot; weakness of the muscles, resembling paralysis and sometimes mistaken for it in severe cases; reflex irritability, rendering rachitic patients liable to laryngismus and tetany; undue perspiration; anæmia and proneness to catarrhal inflammation; and certain anatomical changes in the spleen and liver in aggravated forms of the disease. These many and divers anatomical and functional characters indicate the constitutional or general nature of rachitis. Therefore theories which restrict rachitis to the osseous system are inadequate and erroneous.

Rachitis is probably an ancient disease. It is said that an old statue of Æsop, who was thrown from a precipice by the indignant Delphians 564 years before Christ, exhibited rachitic deformities; and Hippocrates, born 460 years before Christ, is believed to have alluded to it in his treatise on the Articulations.

Occasionally expressions in the works of Celsus and Galen in the second century of the Christian era have led writers on rickets to believe that they also had observed the deformities produced by this disease. But rickets was first investigated in a scientific manner by Whistler, Glisson, and their contemporaries in the middle of the seventeenth century. During the last few years many excellent monographs have been written on this malady, and its causation, pathology, and treatment are better understood than formerly.

Frequency.—Rachitis is a widespread disease, but it is comparatively infrequent in rural localities, where families enjoy the hygienic requirements of pure air, sunlight, and a plentiful diet of good quality. It is most common in crowded and badly-fed families in city tenement-houses, where antihygienic conditions prevail.

Mild cases of rickets, not manifested by any prominent signs or symptoms, are often overlooked, so that the physician is not summoned, or, if he be summoned and have not given particular attention to this disease, he, in not a few instances, does not detect its presence. In the absence of deformity, which occurs later, the fretfulness, tenderness of surface, and perspirations are likely

to be attributed to other causes than the correct one. Hence, according to my observations, rachitis is more common in its milder forms in the asylums and dispensaries and in the tenement-houses of New York, and probably in other American cities, than is commonly believed by the laity, and even by physicians who have given little attention to the disease. A few years since in one of the New York asylums my attention was directed to a rachitic child in whom the anatomical characters of rachitis had become so pronounced that they attracted the attention of the nurses. Prompted by the occurrence of this case, which had developed during my attendance in the asylum, I made an examination of all the infants, and found, what I had previously not suspected, that about one in nine presented unmistakable signs of rachitis, though in a mild form and for the most part in its commencement. The late Dr. John S. Parry of Philadelphia stated that at least 28 per cent. of the children between the ages of one month and five years who came under his observation in the Philadelphia Hospital, during the three years preceding the publication of his paper in 1872, were rachitic. According to Dr. Gee, whose observations were, however, made as far back as 1867 and 1868, of the patients under the age of two years in the London Hospital for Sick Children, 30.3 per cent. were rachitic; and Ritter von Rittershain, whose observations were also made several years ago, stated that of 1623 out-door patients under the age of five years brought to the Clinique at Prague, 504, or 31.1 per cent., manifested this disease. Recently Prof. Henoch of the University of Berlin has stated that he had seen many thousand cases of rachitis, and he adds that its spread in the large cities of Northern and Middle Europe is enormous. He states that his observations in regard to the frequency of rachitis in dispensary practice correspond with those of Ritter, as many as 31 per cent. being rachitic. In Manchester also, with its large number of operatives, Ritchie's statistics show that of 728 out-door patients 219 were rachitic. The curator of the New York Foundling Asylum for the last ten years informs me that he believes, without the accuracy of statistics, that as many as 20 per cent of the cadavers examined by him in the dead-house have presented the anatomical characters of rachitis, usually in a mild form.

The recent large emigration from Europe of destitute families, living from choice or necessity in filth and degradation, who for the most part remain in the cities, occupy small, dark, and dirty apartments, and whose food is of the poorest quality and often insufficient, greatly increases the number of rachitic children in New York and probably in other American cities. In the out-door department of Bellevue, to which many thousand immigrants from the lowest class of European society carry their sick children for treatment, rachitis is not infrequent; and the fact has been observed in this institution that a larger proportion of severe cases attended by marked deformities occur in the Italian families than in those from other parts of Europe. In families of American parentage it is generally admitted that rachitis is more prevalent in the negro than in the white race.

Although this disease occurs most frequently in the families of the destitute and poorly fed, nevertheless children of well-to-do families occasionally suffer from it, even in an aggravated form, in consequence, I think, usually of ignorance on the part of parents in regard to the dietetic requirements of young children. Merei, in his treatise on the Disorders of Infantile Development (London, 1850), states that in Manchester, where his observations were made, one child in every five in comfortable circumstances presented rachitic symptoms. In the United States rachitis is rare in well-to-do families, who provide sufficient and suitable diet for their children and have a proper regard for sani-

tary requirements. When it does occur in such, it is due usually. I think, to improper feeding. But this cause will be discussed in another place.

Diagnosis.—In preparing statistics relating to rachitis it is obviously important that the diagnosis of mild and incipient cases should be clear and unmistakable. What symptoms and anatomical characters indicate rachitis? The fact that an infant has reached its ninth month without a tooth is regarded by Sir William Jenner as a reliable sign of rachitis. In order to determine to what extent dentition is retarded by rachitis—and retarded dentition may be considered a sign of rachitis—Dr. H. R. Purdy, physician to the Out-door Department of Bellevue Hospital, made the following observations:

TABLE I.—*Showing at what Age 200 Infants exhibiting no Signs of Rachitis cut the First Tooth—cases consecutive.*

3 cut first tooth at 2 months.	28 cut first tooth at 8 months.
14 " " " " 3 "	20 " " " " 9 "
16 " " " " 4 "	14 " " " " 10 "
20 " " " " 5 "	15 " " " " 11 "
24 " " " " 6 "	8 " " " " 12 "
37 " " " " 7 "	1 " " " " 13 "

Of these, 132 were wet-nursed, 68 bottle-fed.

TABLE II.—*Showing at what Age 50 Infants exhibiting one or more Rachitic Symptoms cut the First Tooth—cases consecutive (18 wet-nursed, 32 bottle-fed).*

2 cut first tooth at 4 months.	5 cut first tooth at 8 months.	6 cut first tooth at 13 months.
2 " " " " 5 "	6 " " " " 9 "	3 " " " " 14 "
3 " " " " 6 "	7 " " " " 11 "	1 " " " " 16 "
2 " " " " 7 "	5 " " " " 12 "	1 " " " " 18 "

TABLE III.—*Thirty Infants without Teeth, but with pronounced Rachitic Symptoms.* (In all these cases the rachitic rosary, enlarged subcutaneous veins, profuse perspirations, abdominal distention, and enlarged joints were present. Bottle-fed, 21; wet-nursed, 9. Age at which they cut the first tooth.)

6 at 7 months.	1 at 10 months.	2 at 13 months.
10 " 8 "	4 " 11 "	2 " 14 "
1 " 9 "	3 " 12 "	1 " 15 "

It is evident from these interesting statistics that dentition delayed until the ninth, or even the tenth or eleventh month, is not a certain sign of rachitis, but slow teething is common in the rachitic, and therefore it aids in the diagnosis. It is one of the diagnostic signs.

In order to determine whether rachitis incipient or of a mild form be present, all the signs which characterize it should be considered—the fretfulness, free perspiration upon the head, neck, face, and chest, the tenderness of surface, anæmia and general deterioration of health, delayed dentition, swelling of the joints, craniotabes, bending of the long bones, rachitic rosary, misshapen head, prominent frontal and parietal bones, deformity of the thorax with depression of the ribs, projecting or misshapen sternum and prominent abdomen, with Harrison's groove. All these signs and symptoms must be considered before making a diagnosis in incipient or mild rachitis. In order to determine the diagnostic value of enlargement of the costo-chondral articulations, "the rachitic rosary," in three of the New York institutions I have examined these joints in children supposed to be healthy or suffering from other ailments than

rachitis. In many young children believed to be healthy these joints are not appreciable on palpation. In others a slight prominence can be felt in one or more joints. In order that the beading of these articulations be sufficient to indicate rachitis, it should, I think, be plainly detected by the fingers in most of these articulations. Less than this I would not regard as sufficient evidence of this disease.

Age of Occurrence.—Rachitis is, with few exceptions, a disease of infancy. A large majority of the cases occur before the age of three years. Now and then it occurs in the foetus, producing deformities such as are present in typical cases. In the Kinderspital Museum at Prague is a specimen of foetal rachitis described by Ritter. Hink and Winkler also relate foetal cases, and Virchow alludes to a specimen in the Würzburg Museum which exhibits such deformities as characterize rachitis. Bednar even regards foetal rachitis as not uncommon (Hillier, Parry). In the Wood Museum of Bellevue Hospital is a skeleton which is probably similar to those in the Prague and Würzburg museums. It shows in a striking manner the deformities of congenital rachitis. The case occurred in my practice, and the dissection was made by Prof. Francis Delafield. The infant, born at term, died a few hours after birth from atelectasis, apparently produced by the lateral depression of the ribs and contracted state of the thorax. The parents were hard-working English people, whose mode of life and surroundings were such as are known to conduce to rachitis. They were free from syphilitic taint. The accompanying wood-cut represents this skeleton.

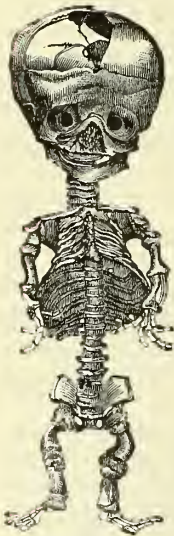
The following remarkable case of supposed foetal rachitis was related to me by Dr. Heitzmann, whose interesting experiments will presently be detailed:

A woman who had frequently inhaled the vapor of lactic acid each day for many months, as she was employed to feed animals with this agent, gave birth to an infant at term which died immediately after it was born. It exhibited the signs of congenital rachitis in a high degree. The skull-bones were completely absent; in the cartilages of the bones of the extremities and in those of the ribs there were scanty depositions of lime salts and numerous infarctions. The death of the child was evidently due to the absence of the skull-bones, inasmuch as the pressure upon the head occurring during birth had caused cerebral hæmorrhage. The organs of the chest and abdomen were fully developed and normal. In the *New York Journal of Obstetrics* for Nov., 1870, Dr. A. Jacobi also published the description of a case of congenital rachitic craniotabes.

Enlargement of the costo-chondral articulations, known as the rachitic rosary, has been observed, though rarely, in infants only a few weeks old. Dr. Parry saw it as early as the sixth week after birth, and Dr. Lee at the third or fourth week. The significance of this enlargement as a sign of rachitis we have treated of elsewhere. We have stated that with few exceptions rachitis begins before the close of the third year. Though first detected and diagnosed at a

later date, it will ordinarily be ascertained, on inquiry, that its symptoms had an earlier beginning. Still, according to certain observers, it may have a considerably later commencement. Glisson, Portal, and Tripier state that they have seen it commence in children who were well on toward the age of puberty. Sir William Jenner says that he has seen children of seven and eight years who were only beginning to suffer from rachitis.

FIG. 1.



Congenital Rachitis.

The following are the aggregate statistics of Bruennische, Von Rittershain, and Ritsche relating to the age at which rachitis occurs :

	No. of Cases.
During the first half year	99
“ “ second half of first year	259
“ “ “ year	342
“ “ “ third year	134
“ “ “ fourth year	31
“ “ “ fifth year	17
Between the fifth and ninth years	21
Aggregate	903

Etiology.—*Inheritance.*—Some patients with rachitis appear to have inherited a predisposition to it. Feeble digestion and defective assimilation in the infant—which are, as we will see, important factors in producing the rachitic state—are often traceable to disease or cachexia of one or both parents. Among the parental causes may be mentioned poverty, hardships, and defective nutrition of either parent; age of father and exhausting discharges of the mother, such as purulent, hæmorrhoidal, or uterine fluxes. The offspring of a tubercular, syphilitic, or otherwise enfeebled parent is more likely to become rachitic than is one of healthy and robust ancestry. We will especially emphasize the syphilitic dyscrasia in either parent as a potent cause, but M. T. Parrot, in his thesis published in 1872, evidently went too far in attempting to show that congenital syphilis is the common cause of rachitis. Most rachitic cases are entirely free from the syphilitic taint, and a large proportion of the children who have inherited the syphilitic dyscrasia do not exhibit any signs of rachitis.

Antihygienic Conditions.—In the damp, dark, filthy, and overcrowded tenement-houses of the city, rickets occurs most frequently and in its severest forms. There can be no doubt that general mal-hygiene is a potent factor in causing this disease, and that it sometimes produces it in those who have inherited good constitutions. On the other hand, many children with healthy parentage and vigorous at birth, reduced by poverty to a life of squalor and privation, do not become rachitic.

Food.—Of the antihygienic conditions which give rise to rachitis, the most common and potent appears to be the use of food not sufficiently nutritious, or, if nutritious, not suited to the age and digestive powers of the child. The use of thin and poor breast-milk and artificial food of poor quality or not suitable for the stage of growth and development is a common cause of rachitis. Those children who have been prematurely weaned, and who have been given a food which is not a proper substitute for the natural aliment, and those too long wet-nursed by scantily-fed and poorly-nourished mothers, and not allowed the additional aliment which they require, are especially liable to this disease. Those children whose digestive power is feeble, from whatever cause, are more likely to become rachitic than those who in a state of robust health have a hearty digestion. Hence we meet with rickets as a sequel of various protracted and exhausting maladies during infancy.

I might relate cases of rachitis occurring during the use of certain of the popular proprietary or commercial foods. I have examined the analyses of these foods made by Prof. Leeds in order to determine what ingredient is lacking, and they are found to contain a considerably smaller percentage of fat than occurs in human milk. Too little fat in the food may, as Cheadle observes, be one of the chief dietetic causes of rachitis. Infants suckled by healthy mothers or wet-nurses who have an abundance of milk, of good quality, do not become rachitic as long as their nutriment is derived from this source. But

those prematurely weaned and given a diet deficient in nutritive properties, and those who are allowed the promiscuous food of the table or have largely a farinaceous diet during the first and second years, when the food should be chiefly milk, are especially liable to become rachitic.

It is an interesting fact, and one that throws light on the dietetic cause of rachitis, that it does not occur in Japan. Physicians who have had abundant opportunities to observe the diseases of the Japanese state that they have never seen or heard of a case among them. M. Remy, in his *Notes Médicales sur le Japon*, says that the Japanese women have a remarkable abundance of milk, and that they suckle their young until the age of five or six years, but their children are also given artificial food after the first year. Remy's explanation of the immunity of the Japanese from rachitis is as follows: "The Japanese have always eaten plentifully of fats and oil of fishes, the blubber of the whale, the eel and loach especially. . . . The universal use of the food under notice from the time of ancient Buddhist flesh-prohibition, but especially the consumption of fish by the lactating women, together with the fish given to the children as supplementary feeding, which at that time is allowed them by Japanese tradition, are, in my opinion, main causes of the non-existence of rachitis in Japan."

Observations on the feeding of animals have also aided in the elucidation of the causation of rachitis. Guérin gave certain puppies a diet of meat four or five months, and they became markedly rachitic, while other puppies of the same litter, suckled by their mother, remained well. At a meeting of the section of Diseases of Children of the British Medical Association, held in August, 1888, Dr. W. B. Cheadle read an instructive paper on rachitis, in which he said that the results of feeding young animals in the Zoological Gardens strongly support the view that a deficiency of animal fats and earthy salts are the most efficient agents in producing rickets. He states that in the Zoological Gardens the young monkeys taken from their mothers and fed with a vegetable diet, chiefly fruits, became rachitic. Such diet is destitute of animal fat, and is deficient in proteids and earthy salts. Two young bears were fed with rice biscuits, and occasionally with lean meat, which they licked, but rarely ate. Fat, proteids, and lime salts were practically excluded from their food. The bears died of extreme rickets while still young. Cheadle also states that more than twenty litters of lions had died successively of rachitis, and the next brood were fed with cod-liver oil, pulverized bones, and milk. In three months all signs of rickets had disappeared. The addition of fat and bone-salts caused the change, and after eighteen months, when the last observations were made, the brood of young lions were strong and healthy. They had received in every respect the same treatment as the litters that had perished, except as regards the diet. The latter had been fed with the carcasses of old horses, which are destitute of fat and whose bones resisted the lions' teeth.

The theory that lactic acid is the causal agent in rachitis has been strongly advocated by Dr. C. Heitzmann, formerly of Vienna, but now of New York. He administered lactic acid by mouth and subcutaneous injection to five dogs, seven cats, two rabbits, and one squirrel. The lactic acid administered to the dogs and cats, with "restricted administration of calcareous food," produced the characteristic enlargement of the epiphyses, and finally the "curvatures of the bones of the extremities." After four or five months of administration of lactic acid the long bones were very flexible, and repeated inflammations of the conjunctiva, bronchi, stomach, and intestines had occurred.

But in many cases of rachitis there is no evidence of an excess of lactic acid, and an objection to the lactic-acid theory apparently valid is that lactic

acid, produced by imperfect digestion, would unite with a base, either the soda or potash in the blood, which is always alkaline, before it reached the osseous system. The more the causation of rachitis is elucidated by observations on man and experiments on animals, the stronger is the evidence that its chief cause is dietetic—that there is a failure to receive or to digest and assimilate certain important substances in the food, particularly the fat, phosphate of lime, and proteids. The deprivation of these alimentary substances produces the rachitic dyscrasia, which is manifested by malnutrition in many tissues. Of course general antihygienic conditions, which lower the vitality, may, as we have stated elsewhere, be a factor in causing rachitis.

Pathology.—Distinguished pathologists and clinical observers who have investigated rachitis, and whose investigations have been chiefly, if not entirely, restricted to the osseous system, have regarded this disease as an inflammation affecting the bones and cartilages. Among those who have expressed this opinion may be mentioned Virchow and Niemeyer. Niemeyer says: “It seems to me that the most probable hypothesis regarding the cause of rachitis is that which refers it to inflammation of the epiphyseal cartilages and periosteum.” The increased vascularity of the periosteum, the proliferation of periosteum and cartilage, the tenderness and pain on motion, and the elevation of temperature in acute forms of the disease, indicate inflammation rather than any other recognized pathological state. If the rachitic disease of the osseous system be regarded as an inflammation, it obviously presents a subacute or chronic character, like cirrhosis and certain forms of chronic nephritis, in which proliferation of connective tissue and sclerosis occur. The eburnation, instead of normal ossification, which terminates the rachitic process, might be considered an osteosclerosis. Moreover, the thickening, hyperæmia, and infiltration of the periosteum, exudation and formation of new vessels in the periosteum and underlying cartilaginous and osseous tissues, are conformable with the theory of the inflammatory nature of rachitis. On the other hand, some of the structural changes in the soft tissues in rachitis which are described in this paper are not such as ordinarily result from inflammatory processes. Billroth, seeing the difficulties in the way of the inflammatory theory, wrote of rachitis that it “cannot be exactly classed among the chronic inflammations, although nearest related to this process.” It seems most in consonance with the facts to regard rachitis as a constitutional or general disease, a dyscrasia affecting the nutrition of various tissues of the body, and producing disease in the osseous system which is either inflammatory or closely allied to inflammation.

CHANGES IN THE SOFT TISSUES.—We have stated that although the conspicuous lesions of rachitis pertain to the skeleton, the soft tissues are also more or less implicated, as might be expected, since the disease is systemic in its nature. The skin in mild cases is but little involved, but as a rule the perspiration of the rachitic is excessive from the head, face, neck, and chest. This may occur before changes are observed in the skeleton. Pyrexia is in some patients absent or slight, but catarrhs of the mucous surfaces are common, and these are likely to give rise to some elevation of temperature. The fever that frequently accompanies severe cases may sometimes result from the disease of the skeleton. In protracted and severe cases the patients become markedly anæmic, but in recent and mild cases the pallor may be so slight as not to attract attention. Emaciation is not pronounced, as a rule, in the rachitic, but in certain patients the muscles throughout the system become shrunk and flabby, partly perhaps in consequence of the gastro-intestinal disorder, indigestion, and malnutrition, partly perhaps from want of use, for the rachitic are likely to be passive.

Mucous Membranes.—Rachitis, as we have stated above, increases the liability to catarrh of the mucous surfaces. Writers on this disease have remarked the frequent occurrence of bronchitis, broncho-pneumonia, enterocolitis, and conjunctivitis.

Ligaments.—The ligaments become relaxed and flabby, giving unusual mobility to the joints and unsteadiness to the movements. The fibrous bands which unite the vertebræ, as well as the ligaments of the extremities, participate in the relaxation. Talipes valgus and knock-knee are especially likely to occur in rickets as a result of the relaxation of ligaments, even when the bones are but slightly involved. Kyphosis, lordosis, and scoliosis—backward, forward, and lateral curvatures of the spine—also result from relaxation of the ligaments, aided by the softening and change in shape of vertebræ and of the intervertebral cartilages.

The Spleen and Liver.—The spleen is sometimes enlarged, as ascertained by palpation and percussion. Ritter von Rittershain found this organ decidedly enlarged in 10 out of 35 cases which he examined after death. The enlargement is the result of cellular proliferation, common in diseases which are attended by a dyscrasia. In a recent very anæmic and fatal case of rachitis in the New York Foundling Asylum the spleen extended below the level of the umbilicus. But in many cases of rachitis, even when profound, splenic enlargement is slight or is not appreciable.

The liver in many patients undergoes no perceptible change, except that it is carried downward by the lateral depression of the ribs. It is occasionally enlarged from fatty infiltration, but no special significance attaches to this, for fatty liver is common in various forms of disease attended by innutrition and wasting. It is common in tuberculosis and in protracted intestinal catarrh, and its pathological significance appears to be the same in these various diseases. There can be no doubt that Sir William Jenner errs when he states that albuminoid infiltration of the liver is common in rachitis. Parry, Gee, Dickinson, and Senator agree that it is rare, and that when it does occur it is a coincidence.

In the discussion of rickets at the meeting of the British Medical Association in August, 1888, Dr. Ranke of Munich said that, according to the records of 34 post-mortem examinations of rachitic cases in Virchow's Pathological Institute between 1872 and 1880, 13 exhibited changes in the liver, mostly parenchymatous fatty infiltration with increase of volume. In the 34 cases the spleen was recorded enlarged in 9 and small in 2. In the remaining 23 cases the size and appearance of the spleen were probably normal, or some mention would have been made of it. Dr. Ranke also consulted the records of the Munich Pathological Institute under Professor Bollinger, and in 9 of 25 post-mortem examinations of rachitic cases more or less enlargement of the liver was recorded. We may therefore infer from these carefully conducted examinations that enlargement and structural changes of the liver and spleen only occasionally occur in rachitis—that in the majority of cases this disease runs its course without any notable alteration in these organs. My own observations lead me to believe that hypertrophy of the spleen, and probably also of the liver, occurs chiefly in decidedly anæmic subjects.

The Abdomen is Protuberant from various causes. The lateral depression of the thoracic walls causes the liver and spleen to descend a little lower in the abdominal cavity than natural, producing at the base of the chest anteriorly Harrison's groove, which is transverse and corresponds with the insertion of the diaphragm. The enlargement of the liver and spleen, the feeble tonicity of the intestinal muscular fibres, and consequent distention of the intestines with gas, and the rachitic shortening of the spinal column,

which causes approximation of the ribs and pelvis, necessarily produce abdominal protuberance.

The Kidneys and Urine.—Observations thus far have not detected any structural change or disease of the kidneys attributable to rachitis, except that this organ is enlarged in some cases. Moreover, the records of the urine are so conflicting that more exact and more numerous examinations of this excretion are required before any positive statement can be made in reference to its composition. Dr. C. H. Flagge has seen two cases in which there were large quantities of uric acid in the urine. Ephraim also mentions an increased elimination of uric acid up to 18 per cent. Ephraim likewise, as well as Marchand and Lehmann, state that there is an increase of phosphate of lime and the occurrence of lactic acid in the urine.

Brain and Spinal Cord.—It is not improbable that the symptoms of rachitis which are referable to the nervous system, such as laryngismus stridulus, tetany, convulsions, and weakness or paralysis of the extremities, may be largely due to the pressure exerted in places upon the cerebro-spinal axis by its bony covering. Hence we will postpone their consideration until we have described the changes produced by rachitis in the osseous system.

CHANGES IN THE OSSEOUS SYSTEM.—A knowledge of the normal anatomy and normal development of the osseous system will enable us to better understand the changes which occur in this system in disease, and especially, which concerns us at present, in rachitis. Hence we will give a brief résumé of the anatomy of the skeleton in health before we consider the changes produced in it by rachitis.

Osseous System in Health.—In health and when fully developed, bone consists of animal matter (chiefly gelatin) and earthy salts, in the proportion, by weight, of about one part of the former to two of the latter. The following is the analysis, which may be regarded as approximately correct, of healthy human bone of the adult:

	Animal matter	33.30
	Tribasic phosphate of calcium	51.04
	Carbonate of calcium	11.30
Earthy salts. {	Fluoride of calcium	2.00
	Phosphate of magnesium	1.16
	Soda and chloride of sodium	1.20
		100.00

In childhood the bones are softer, more elastic, and less likely to fracture than in the adult. Of the earthy salts in bone, it is seen that the phosphate of calcium is the most abundant, and it is the most important. Hence it is termed "bone earth." The phosphate of calcium, combined with animal matter, produces a hard compound. The enamel of the tooth consists chiefly of phosphate of calcium ($88\frac{1}{2}$ per cent.), while the softer egg-shell consists chiefly of the carbonate of calcium. The strength of bone is remarkable, being, according to Holden, when compared with wood, nearly three times that of the elm or ash, and double that of the oak. It is elastic on account of the animal matter which it contains. If a long bone be placed at right angles upon a hard substance, and the projecting end receive a blow from a hammer, the latter will rebound. The Arab children are said to make bows of the camel's ribs.

If a longitudinal section be made through a long bone, we observe a hard or compact outer part, and in the interior the medullary canal, containing marrow. In birds of flight the hollow of the bones contains air instead of marrow, and this air communicates with the lungs.

The hard or compact portion of bone, though solid like a stone, consists of layers in close apposition, so that there is no interval between them. On approaching the joints the internal layers of the compact structure separate from each other, forming the cancellous tissue, so that the compact wall becomes thinner. If the earthy salts be removed by an acid, the animal matter remaining is found to consist of layers, which can be separated from each other. In inflammation the afflux of blood and the exudation cause separation of the layers and enlargement of the bone.

The cancellous tissue occupies the interior of the bone, and is most abundant in its articular ends. The bony layers in the cancellous structure are separated from each other, so as to form cavities, which are strengthened by cross-plates like latticework. In the adult the marrow in the interior of the shafts of the long bones is yellow, consisting of 96 per cent. of fat, but in the articular ends of the long bones, in the ribs, cranial bones, and short bones, the marrow has a reddish tinge, and it consists of about 75 per cent. of water and about 25 per cent. of albumin, without fat or only a trace of it. This kind of marrow occurs in all the bones of the fœtus and the infant, and it contains cells with many nuclei, designated "myeloid cells." Holden says that bones are as minutely provided with blood-vessels and nerves as are the soft tissues. Near the extremities of the long bones are numerous minute openings through which blood is conveyed to and from the cancellous tissue. On the shafts of the long bones are slight grooves parallel with the shafts, at the bottom of which are minute holes, scarcely visible, through which blood is conveyed to and from the compact tissue. The blood which supplies the osseous tissue is conveyed through these holes by minute arteries from the vessels of the periosteum, and is returned by veins to the periosteum. Near the middle of the shaft of the long bone is a distinct canal passing obliquely through the shaft. This canal contains the nutrient artery of the medulla, dividing, after entering the medullary cavity, into two branches, one passing upward and the other downward. The blood-vessels supplying the different parts of the bone from these various sources intercommunicate. Other bones than the long bones are supplied with blood in a similar manner, and the nutrient vessels are accompanied by nerves, as in other parts of the system.

The microscope is required in order to reveal the minute anatomy of bone. It is found to consist of canals, termed the Haversian, and around each canal the bone is arranged in concentric layers, like the concentric rings of a tree. Between the rings are dark spots, designated lacunæ, arranged concentrically, now known to be minute reservoirs containing blood. Minute lines are seen connecting the reservoirs with each other and with the adjacent Haversian canal. The lines are minute blood-vessels, and through them the blood is conveyed to every part of the bone. They are designated canaliculi. They connect externally with the vessels of the periosteum, and internally with the vessels of the medullary membrane or endosteum. In the interspaces between the lacunæ and canaliculi, in the animal matter, an infinite number of osseous granules is deposited, consisting mainly of phosphate and carbonate of lime.

Alterations in the Osseous System in Rachitis.—For convenience of description the course of rachitis as regards the osseous system is divided into three periods: (1) That of proliferation and altered nutrition of cartilage and periosteum; (2) That of curvature and deformity; (3) That of reconstruction.

1. **Anatomical Characters in the Stage of Proliferation and Altered Nutrition.**—The long bones in normal growth increase in length by the formation of bone in the cartilage between the diaphysis and epiphysis, and

in thickness by the development of bone from the vascular and cellular under-surface of the periosteum. As regards the flat and short bones, growth in the thickness occurs from the periosteum, and growth in breadth occurs from the development and ossification of the cartilaginous borders and edges, which correspond with the epiphyseal cartilage of the long bones.

If we examine the epiphyseal cartilage of a long bone during normal ossification, we observe, beginning at the distal end, a white zone, consisting of the hyaline matrix, in which are the usual cartilage-cells. This constitutes most of the cartilage. Underneath this, and nearer the bone, is the zone of proliferation, the cartilage in which is softer and more yielding than that of the distal zone, in consequence of cell-formation and absorption of the matrix to make way for cell-groups. Each cell in the proliferating zone has divided into two cells, and each of these cells into two other cells; and the division has been repeated, so that eight cells instead of one are observed, surrounded by a common capsule. The capsule becomes distended by the cell-multiplication and the swelling of each cell, the size of which is considerably greater than that of the parent cell. Near the bone, along the extremity of the diaphysis, the cell-groups, enclosed in their capsules, nearly touch each other, the matrix having been for the most part absorbed. The end of the diaphysis is covered with a layer of these cell-groups about to undergo ossification, with almost no intervening matrix. The proliferating zone has very little depth. It appears to the naked eye as a very thin, scarcely perceptible layer of a reddish-gray color upon the end of the shaft. It is so thin that it but slightly increases the thickness of the cartilage.

In rachitis the state is different. The zone of proliferation, instead of being confined to a single or at most double layer of cell-groups, consists of many layers, involving nearly the whole epiphyseal cartilage. The cells, still enclosed in their capsules, undergo a more frequent division than in health, so that, instead of groups of eight cells, as in the normal state, each group consists of thirty or forty cells enclosed in the distended capsule. Therefore in rachitis the proliferating cartilaginous zone is a broad cushion, very soft, of a grayish translucent appearance, causing the characteristic swelling observed around the joint. Over the distal end of the proliferating cartilage there may still be a zone, though perhaps of little depth, of normal cartilage like that in health.

While the changes described above occur in the cartilages, the ossifying process is arrested or rendered abnormal. We indeed perceive an effort in the direction of bone-formation. The Haversian canals, surrounded by capillary loops, extend from the bone into the proliferating zone of cartilage. Their extension is effected by absorption of the matrix and appropriation of cell-groups which lie in their way. The cells in these groups, as they enter the Haversian system, become much smaller by rapid segmentation, forming medullary cells. We also find, as further evidence of the attempt at bone-formation, granules and masses of lime scattered through the cartilage, and here and there spiculæ and nodules of true bone springing up from the bony substance of the shaft. Some of the canals are prolonged far into the cartilage—nearly, indeed, to its free surface—but most of them terminate in its lowest portions.

We have stated that the growth of bone in thickness occurs from the under surface of the periosteum. In health a soft, vascular germinal tissue springs from the periosteal surface, rapidly receives lime salts, and is transformed into bone. This germinal tissue, consisting largely of capillaries rising from the fibrous tissue of the periosteum, is a very thin substance, barely visible, transient, and constantly changing from its conversion into bone.

In rachitis this vascular subperiosteal tissue, not undergoing, or undergoing slowly and imperfectly, the osseous transformation, and at the same time increasing more rapidly than in health under the irritating influence of the rachitic disease, becomes a thick layer. Its color and appearance are like spleen-pulp, so that the older observers supposed that there was hæmorrhagic extravasation between the periosteum and the bone. There is, however, no extravasation of blood, unless it accidentally occurs from the numerous delicate capillaries. The resemblance to extravasated blood or spleen-pulp is due to the abundant growth of large and thin-walled capillaries from the under surface of the periosteum, as shown by the microscope. This vascular outgrowth is, for the most part, quite uniform over the shafts of the long bones, while upon the cranial bones its thickness is much greater in one locality than in another. The attempt at ossification also appears in this tissue. Lime salts are scantily and loosely deposited through it, forming osteophytes, vascular and fragile, rather than true bone. The question naturally arises, How does rachitis affect bone which is already formed when the rachitic state begins? Virchow's answer is the following: "Rachitis has by more accurate investigation been shown to consist, not in a process of softening in the old bone, as it has previously been considered to be, but in a non-consolidation of the fresh layers as they form: the old layers being consumed by the normally progressive formation of medullary cavities, and the new remaining soft, the bone becomes brittle."

We have seen that in healthy bone the earthy salts are in excess of organic matter nearly in the proportion of two to one, but in rachitis the proportion is reversed, the organic matter being much in excess. The following table gives analyses of rachitic bones by Marchand, Davy, Boettger, and Friedleben:

	Femur.		Radius.		Vertebra.	
	Inorganic.	Organic.	Inorganic.	Organic.	Inorganic.	Organic.
Case I. . . .	20.60	74.40	21.24	78.76	18.68	81.32
Case II. . . .	37.80	62.20	20.00	80.00	32.29	67.71
Case III. . . .	20.89	79.11				
Case IV. . . .	52.85	47.15				

As might be expected, the relative proportion of the inorganic matter (the earthy salts) and the organic matter varies greatly in different cases. In severe rachitis many bones are affected. It is stated that there is no bone in the entire skeleton that may not suffer, but in mild cases only a few are involved, at least to such an extent as to produce structural changes appreciable to touch or sight.

Rachitic bone, when the disease is still in its active period, presents a bluish or dusky-red appearance from its increased vascularity. After a variable time—weeks or months according to the severity of the disease—deformities begin to appear.

2. Anatomical Characters of the Stage of Deformity.—CHARACTERS OF THE RACHITIC FÆTUS.—Spiegelberg's description of the rachitic fœtus corresponds for the most part with what I observed in the one whose skeleton is represented in a foregoing page. According to this writer, the body and limbs are plump, the latter short and curved, the abdomen large and prominent, and the head sometimes hydrocephalic. The skin is well developed and movable, the adipose tissue sufficient, the liver large, the epiphyses swollen and soft, the short and curved diaphyses sometimes broken; the rotundity of the thorax is preserved, and the sternum is not carried forward, since there has been no respiration. The ribs in softness and liability to fracture correspond with the long bones of the extremities. The sternum, most of all the bones,

PLATE XI.



RACHITIS.

shows the delay in ossification; the clavicle is among those least affected. The cranium may be represented by a membranous bag with plaques of bone, or the cranial bones may be formed and in shape, but thickened and softened; the sacral promontory is pressed-forward and downward; the ilia flattened and widened; the pubic arch increased.

CHARACTERS OF THE RACHITIC CHILD.—In typical rachitis the bone seldom retains its normal form or shape: its projecting points are rounded, and as soon as it softens it begins to yield to pressure exerted upon it. Hence the curvatures so common and characteristic. The portion of a long bone which is formed after rachitis commences contains so little earthy matter that it bends readily in its fresh state either by muscular action or by the weight of the trunk “in the manner,” says Vogel, “of a quill or willow stick.” The interior of the bone, which was formed before rachitis began, and which contains nearly or quite the normal proportion of lime, is likely to break instead of bending, but, as it is surrounded on all sides by the soft tissue, the fragments are not displaced, and probably do not crepitate. So scanty is the calcareous deposition in typical cases that, says Tröusseau, “the bones . . . can be cut with a knife with as much ease as a carrot or other soft root,” and the dried specimen weighs from one-sixth to one-eighth of the weight of normal bone. One writer states that the dried rachitic bone is sometimes so porous from the small amount of lime which it contains that it is possible to respire through it as through a sponge.

In ordinary cases the bones which exhibit most strikingly the rachitic change, and which, therefore, should be examined carefully in making the diagnosis, are the cranial bones, the ribs, and the radius—the sternal ends of the ribs and the lower end of the radius. It is seldom that these bones do not give evidence of the disease if it be present, and in greater degree than other bones. They are the first to be affected to an extent that is appreciable to the observer.

Changes in the Cranial Bones.—In these bones interesting and important alterations occur. Their edges, which correspond with the epiphyseal cartilages of long bones, undergo proliferation, and become thickened like the latter. This thickening and the delayed union of the sutures produce grooves which can be traced by the fingers between the bones, and which are sometimes appreciable to the sight. Rachitis causes enlargement of the cranium, but the enlargement seems greater than it really is, on account of the retarded growth of the facial bones. In a discussion on rachitis in the London Pathological Society, reported in the *London Lancet* (1880, ii, 1017), it was stated that in seventeen rachitic children with an average age of 4.72 years, the average circumference of the head was 21.22 inches, while in the same number who were non-rachitic, and whose average age was 6.05 years, the average circumference was 19.95 inches. The retarded ossification is manifested not only in the open sutures, but also in the large size and patency of the fontanelles, which are not closed until long after the usual time. The anterior fontanelle in the healthy infant is closed at about the fifteenth or sixteenth month, but in the rachitic it remains membranous a longer time: in some cases it is still membranous as late as the third or fourth year. Since examination of the anterior fontanelle aids in determining whether or not rachitis be present, it should be borne in mind that in the normal state this space increases in size till the seventh month, when it is at its maximum, and that after the ninth month it becomes progressively smaller. Ossification in severe rachitis is retarded for a longer period than is stated above, for Gerhard relates a case in which the anterior fontanelle had not entirely closed at the ninth year.

The shape of the rachitic head varies. In general, instead of its normal rounded form it approaches a square shape. Another type is sometimes observed in which there is no marked angularity, but in which the antero-posterior diameter is enlarged. In the square head the forehead projects, and both the frontal and parietal protuberances are unusually prominent. The sutures are depressed to a certain extent, as has already been mentioned, and the anterior, lateral, superior, and posterior surfaces are more flattened than in health. The undue prominence of the frontal and parietal eminences is largely due to the exaggerated proliferation of the periosteum and to the vascularity and infiltration underneath. Enlarged veins are seen ramifying in the scalp, which in marked rachitis supports a scanty growth of hair. The free perspiration from the scalp, and in some cases the activity of its sebaceous follicles, will be mentioned elsewhere.

Craniotabes.—Thinning of the cranial bones in places, so that the brain lacked proper protection, had long been noticed in the examination of rachitic heads, but the injury that resulted to the infant was overlooked until pointed out by Elsässer. Craniotabes occurs for the most part in infants under the age of one year, and a large proportion are under eight months. Its occurrence in the foetus, as shown by a case published in the *New York Obstetrical Journal* in 1870, and by Heitzmann's case, has already been alluded to. The factors in producing this thinning are rachitic softening of the bones and pressure from the brain within and from the pillow without. Consequently, the portions of the cranium in which the thinning is most pronounced are the posterior and lateral, the occipital bone and the posterior half of the parietal. If the infant lie in its crib chiefly on one side, on this side the craniotabes occurs, while those portions of the cranium which are not pressed upon exhibit no thinning or a less degree of it. The soft spots in the cranium are yielding when pressed upon, and in the cadaver they are seen to be translucent when the bone is held to the light. There are in some instances simple depressions like erosions in the bone, a continuous but thin bony layer remaining. In other cases, such as have been particularly examined and studied by physicians, the bony absorption has been complete over areas of greater or less extent. On examining a child for craniotabes it should be borne in mind that the margins of the cranial bones, even when there is no thinning, but thickening from the cartilaginous proliferation, are flexible in the rachitic. The pressure must be made in a direction away from the sutures to ascertain whether craniotabes has occurred. The pressure should at first be made lightly and cautiously with the fingers, for if there be total absence, unless of very little extent, deep and forcible pressure might injure the brain, since so soft and delicate an organ, covered only by scalp and dura mater, badly tolerates pressure. If the first examination detect no soft place, the fingers may be pressed more firmly against the scalp, when, if the bone be much thinned, so that there is only a small layer of lime salts underneath, it will be found to yield. The sensation communicated to the fingers when there is an open space in the cranium, and the dura mater and scalp are in contact, has been likened to that experienced when pressing upon a fully-distended bladder. At a meeting of the London Pathological Society, reported in the *Lancet* for November, 1880, Dr. Lees presented statistics to show that craniotabes is one of the lesions of inherited syphilis; but whether it does sometimes result from inherited syphilis or not, the evidence that there is a cranial softening which is strictly rachitic, and which occurs in those who have not inherited syphilis, appears from reported observations to be conclusive.

Changes in the Vertebrae, etc.—The short bones which participate in the rachitic disease become softer and more yielding, and their cancelli are filled

with a reddish pulpy substance. In many rachitic cases the vertebræ are but slightly involved, so that no deformity of the spinal column results; but occasionally, when many bones are affected, the vertebræ and intervertebral carti-

FIG. 2.

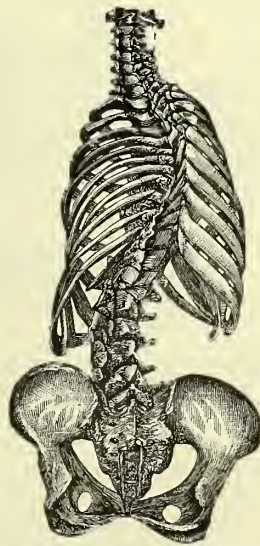


Head of a Rachitic Child in the New York Infant Asylum. This child also had laryngismus stridulus.

lages soften, and spinal curvatures result. The curvatures are due to the weight of the shoulders and head on the spinal column. They are, with some deviations, an exaggeration of those present in the normal state. Rachitic curvatures of the spinal column are therefore mainly antero-posterior, often with more or less lateral deflection. When there is much curvature the vertebræ become wedge-shaped, narrowed upon the concavity and thickened upon the convexity. The intervertebral cartilages are also more or less changed by the pressure, being thinned where the vertebræ approximate to each other on the concave aspect of the curvature, and of normal thickness or thicker than normal upon the convexity. The accompanying wood-cut exhibits the appearance and nature of rachitic spinal curvature continuing into adult life. Rachitis, having occurred at the usual age, resulted in the permanent deformity here illustrated.

In extreme cases, fortunately rare, the functions of important organs may be seriously impaired by the curvature and consequent compression, as they are in Pott's disease. Thus, according to Miller, the aorta has been so doubled upon itself as to materially diminish the flow of blood to the lower extremities, so that their nutrition was sensibly impaired. The effect of so great curvature upon the heart and lungs must obviously be detrimental. At first the spinal curvatures disappear when the child reclines or is lifted by the axillæ so as to raise the head and shoulders from the spine; but when the deformity has continued so long that the vertebræ and cartilages have become

FIG. 3.



Rachitic Spinal Curvature in an Adult (from a specimen in the Wood Museum, Bellevue Hospital).

wedge-shaped, it remains for life or can only be rectified slowly and with difficulty by mechanical appliances. As seen in the wood-cut, the common curvature in the dorsal region is backward (kyphosis), while to compensate the patient instinctively carries the neck forward, with the head thrown back, causing cervical lordosis, a similar anterior curvature being common in the lumbar region. Lateral curvature (scoliosis) may or may not be present even when there is considerable antero-posterior flexure. Scoliosis is sometimes produced by the nurse in carrying the infant habitually over one arm.

Changes in the Maxillæ.—Fleischmann has investigated the changes which rachitis produces in the maxillary bones. Stunted growth of the facial bones, generally, has long been known, and has been remarked upon by various writers; but, according to Fleischmann, other interesting changes occur in the jaw-bones which affect the direction and position of the teeth. According to this observer, the arched shape of the lower jaw becomes polygonal, and the direction of its alveoli also changes, so that they incline inward. This deviation in the arch and in the alveolar border of the lower jaw, which begins in the region of the canine teeth, necessarily causes softening of the jaw. Commencing soon after, a change is observed in the upper jaw-bone from the zygomatic arch forward, so as to cause lengthening of this bone, changing the shape of the arch and the position of the teeth.

FIG. 4.



Rachitic Child with characteristic deformity of head and ribs. (From a patient in the New York Foundling Hospital).

The external incisors, instead of being in front, have a lateral position, and when the jaws are closed the superior incisors and molars overlap the corresponding teeth of the lower jaw in front, and upon the sides—a condition opposite to that seen in the jaws of old people. Fleischmann attributes these changes in the lower jaw to the action of the masseter and the mylo-hyoid muscles, and perhaps the genio-glossus, and to pressure of the lip, the deficiency of earthy salts in the bone rendering it more easily acted on by the muscles. The change in the upper jaw-bone he attributes largely to lateral pressure of the zygomatic arches.

Changes in the Ribs.—The ribs are easily affected in rachitis. The swelling of their anterior ends, where they unite with the costal cartilages, producing the “rachitic rosary,” has been already alluded to as one of the first

and most conspicuous signs of rachitis. The costochondral articulations are enlarged in all directions, appearing as nodules under the skin. If at an autopsy an opportunity of inspecting the pleural surface of the articulation occur, the nodular prominence is seen to be even greater and more distinct than under the skin (Fig. 4).

The deformity of the thorax, consequent upon softening of the ribs, is interesting. Commencing with the spine, the ribs extend nearly directly outward: at the union of the dorsal and lateral portions they make a short curve forward and then turn inward, also with a short curve, toward the sternum

(Fig. 5). This abrupt bending of the ribs, which in their softened state has been caused by atmospheric pressure during respiration, produces a depression in the thoracic wall at about the point where the ribs and their cartilages unite. A groove extends on the antero-lateral aspect of the thorax from the second or third rib downward and a little outward. In some cases the costochondral articulations are in the line of greatest depression in the thoracic walls; in other cases they are a little inside or outside of the deepest part of the groove. The transverse diameter, therefore, of the anterior half of the thorax is less than that in the normal rotund form of health. This necessarily diminishes the antero-lateral expansion of the lungs in inspiration and causes unusual prominence of the sternum. Hence the expressions "pigeon-breasted," "resemblance to the prow of a ship," etc. applied to this deformity. The presence of the heart renders the depression or groove less on the left side between the fourth and sixth ribs than on the opposite side, since this organ affords partial support to the chest-wall. On the other hand, the depression on the right side below the sixth or seventh rib is, on account of the support given by the liver, less than on the left side. But on the left side, as well as on the right, the lower part of the thorax, that below the eighth or ninth ribs, widens, being pressed outward and supported by the abdominal viscera. This gives rise to an antero-lateral furrow or groove near the base of the chest, sometimes designated Harrison's groove.

The ribs with their attached muscles are important agents in respiration, but their soft and yielding nature in the rachitic retards, and to a great

FIG. 5.



Deformity of Chest in Rachitis.

extent prevents, the lateral expansion of the thorax which is necessary for normal and full inspiration. The action of the respiratory muscles and the pressure of the air from within descending along the air-passages is not suffi-

cient to fully overcome the external atmospheric pressure in the absence of the proper resiliency of the ribs. Consequently with each inspiration we observe more or less sinking of the thorax on each side, just as when a moderate obstruction to the entrance of air exists in the larynx or trachea. As the ribs become firmer from the deposit of lime salts, respiration is more regular and normal.

Changes in Bones of Upper Extremities.—Although swelling of the lower end of the radius is one of the earliest signs of rachitis, the bones of the upper extremities are less frequently curved and distorted than those of the lower extremities. The clavicle sometimes softens and bends, producing two curvatures—one backward near the scapula, and another, of larger radius, nearer the sternum, directed forward and a little upward. Careful examination shows, in some rachitic patients, thickening of the margins of the scapulæ like that of the cranial bones. The humerus is occasionally bent, and usually at the insertion of the deltoid in consequence of the powerful action of this muscle in raising and supporting the arm. The radius and ulna are bent outward and twisted. This deformity is attributed by Sir William Jenner to the fact that rickety children support themselves while in the sitting posture upon the palms of the hands pressed upon the floor or couch. Supporting the weight of the body in this manner not only, in his opinion, causes bending of the ulna and radius, but also aids in producing the deformities of the humerus and clavicle.

Changes in the Bones of the Pelvis.—The deformities of the pelvic bones resulting from rachitic softening are very important in the female infant, since pelvic deformities during the procreative period are the common cause of tedious or instrumental labor and stillbirth. These deformities, which elongate some and contract other axes of the pelvis, necessarily occur when the rachitic child is in the erect position, since the pelvic bones support the weight of the trunk, head, and shoulders. A common deformity produced in this manner is the carrying forward of the promontory of the sacrum, which sustains the weight of the spine. There is, moreover, twofold pressure from below—that caused by the heads of the thigh-bones in standing, and that exercised by the tuberosities of the ischia in sitting. Both these forms of pressure have a tendency to narrow the outlet of the pelvis. Hence the marriage of the female who has been rachitic in infancy may involve serious consequences.

FIG. 6.



FIG. 7.

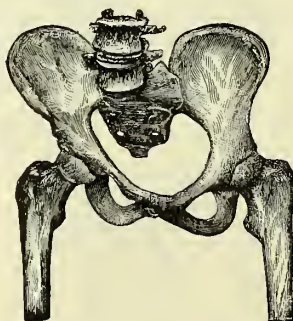
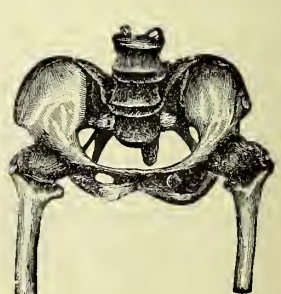


FIG. 8.



Rachitic Deformities of the Pelvis (from specimens in Wood's Museum).

Many of the tedious instrumental labors in the families of the city poor, which severely tax the patience and endurance of young practitioners, are attributable to rickets in early life.

Changes in the Bones of the Lower Extremities.—The curvature of the femur is usually forward or forward and outward. The neck of the femur sometimes bends by the weight of the body or by use of the legs, so that the

FIG. 9.



FIG. 10.



Rachitic Deformities of the Femur (Wood's Museum).

angle which it forms with the shaft is changed. The accompanying wood-cuts show the rachitic bend of this bone in an adult, years after rachitis had ceased and when the bone had become consolidated by the new deposition of lime salts. (Figs. 9 and 10.)

The curvature of the tibia and fibula varies in different cases. In those under the age of one year it is likely to be outward, so that the knees are separated from each other. In those old enough to stand, the weight of the body usually determines a forward bending of these bones. In one case in my practice an anterior curvature, so abrupt that an angle of about 70° was formed, existed about five inches above each ankle. This patient, although old enough to walk, almost constantly sat during the day with the feet extended beyond the sofa, so that the edge of the latter corresponded with the abrupt curvature or angle of the legs. It seemed that the weight of the feet, unsupported beyond the edge of the sofa, had caused these curvatures, especially as the case was one of very marked rachitic softening of the different bones.

Still, tibial and fibular bending at this point has been noticed by different observers, who have attributed it to the weight of the body in walking. Various other curvatures besides those mentioned occur in the bones of the lower extremities, the direction in which the limbs bend being determined by the particular circumstances of the case. In mild cases of rickets most of the deformities described above may be lacking, but in typical cases certain of them stand out prominently, so as to be readily detected by one familiar with the disease. In all such cases the nature of the malady is apparent, for the changes that occur are not only conspicuous, but pathognomonic.

Rachitis produces another important effect on the skeleton. Its growth is stunted, not only during the rachitic period, but subsequently, so that those who have been rachitic in childhood, unless very mildly, have less than the average stature in adult life. The stunted growth is apparent, though ample allowance be made for curvatures. The arrest of development is greater in some bones than in others. It is greatest in the bones of the face, pelvis, and lower extremities. Stunted growth of the pelvic bones of the female infant, conjoined

FIG. 11.



FIG. 12.



Rachitic Deformities of the Femur, Tibia, and Fibula (Wood's Museum).

with the deformities alluded to above, may seriously affect her subsequent life, for the stunted development of the pelvic bones, like the deformities mentioned above, constitutes a valid reason for avoiding marriage. As a rule, the older the child is when rachitis begins, the less is the skeleton affected and the less, consequently, is the deformity.

Effect of Rachitis on Dentition.—As might be expected from the nature of rachitis, dentition suffers severely. The delay in dentition has been considered elsewhere in this paper. Teeth which appear during the rachitic state are frail, deficient in enamel, and crumble readily. They decay and break before the usual time. If certain teeth have appeared before rachitis begins, several months elapse before others cut the gum. It is even said that a child who has rachitis severely for a lengthened period may never have a tooth, and may remain toothless for life; but I have never observed such a case. Ordinarily, when the rachitic state ceases and the health is fully restored dentition goes on in the normal way.

3. Anatomical Characters of the Stage of Reconstruction.—This stage will be better understood if we recollect what has occurred during the first and second stages. The very vascular periosteum is drawn tightly over the convexities, the pressure upon which diminishes the hyperæmia and the amount of exudation underneath. Over the concavities the periosteum is loose: it is hyperæmic with abundant new capillaries, the interspace between it and the bone being filled with the exuded soft material having a gelatiniform appearance. The reparative process goes forward rapidly, the deposition of lime salts being more abundant upon the concave surfaces, where there has been free exudation with no compression of the capillaries, than elsewhere. The lime salts are deposited from the blood. Consequently, from the increased capillary circulation and hyperæmic state of the periosteum produced by rachitis, the earthy material is rapidly deposited wherever there is an open space under the periosteum and where the capillaries are in a state of enlargement. Hence the reconstructed bone is thicker and firmer upon the concave aspect of the long bones than elsewhere, and thinnest upon the convex aspect, where the periosteum is more tense and its capillaries more or less compressed.

Normal ossification does not at first take place during the reparative stage. The deposition of the earthy salts is designated by some writers as a petrification rather than a true bone-formation. Trousseau likens it to the formation of a callus upon a fracture. A deposition occurs of lime salts more compact than ordinary bone. The term "eburnation" has been applied to this new osseous formation, and I have designated it osteo-sclerosis. It resembles, as regards its hardness and morphological appearance, the enamel of the tooth rather than true bone, the Haversian canals and lacunæ being small and imperfectly formed. Of course after complete recovery the subsequent formation of bone is normal. Recovery from rickets is gradual. Little by little the cartilaginous and periosteal proliferations cease, the hyperæmia abates, and the various parts of the osseous system and the soft tissues resume their normal function and development.

General Symptoms of Rachitis.—Preceding and accompanying rachitis symptoms may be present which are due to indigestion and intestinal catarrh, such as flatulence, unhealthy stools, and poor and capricious appetite. When rachitis begins the infant becomes fretful; its sleep is frequently restless and disturbed, and it awakens often. It repels attempts to amuse it, and is apparently annoyed by them. Nurse and mother speak of it as a cross child. It perspires freely from the head and neck both when awake and when asleep, while its extremities and trunk are dry. Its pillow is wet with perspiration during sleep,

and sweat-drops may be seen upon forehead and face. If the surface be dry, a little excitement or elevation of temperature causes perspiration to appear. The rachitic child does not well tolerate the bed-clothes, and it attempts to throw them off from its limbs, even in cool weather lying exposed and causing considerable annoyance to the nurse, who strives to prevent its taking cold. Sometimes miliaria due to the moist state of the skin appears upon the face and neck. We have elsewhere stated that the subcutaneous veins that return blood from the head are large and the jugular veins full. Another symptom is soon observed, to wit: tenderness over a considerable part of the surface, perhaps largely due to the morbid state of the periosteum over so many bones, though it is also experienced when pressure is made upon soft parts, as the abdomen. The tenderness is probably the cause in part of the fretful disposition. The little patient appears to dread to be touched; its flesh is sore; it repels attempts to amuse it, and wishes to be quiet. Dangling it upon the arms, swinging it, or even walking with it, which delights the healthy child and elicits a smile or notes of glee, only adds to its discomfort. It is most at ease when left alone upon a soft cot or pillow, or, if it have craniotabes, when quietly held over the shoulder. Languor, disinclination to use the limbs or to play, moderate thirst, with other symptoms referable to the digestive apparatus which are present in many cases, and which have already been described, are soon followed by changes in the skeleton that are perceptible to the sight and on palpation. The pulse and temperature in a large proportion of the ordinary chronic cases do not deviate from the healthy state, except that in some patients there is a moderate rise in temperature and acceleration of the pulse in the latter part of the day, indicative of a slight fever.

A *bruit de souffle* of greater or less intensity, synchronous with the pulse, has frequently been heard in rachitic cases by applying the ear over the anterior fontanelle. Drs. Whitney and Fischer, New England physicians, first called attention to this murmur, believing it to be a sign of chronic hydrocephalus. MM. Rilliet and Barthez heard it in cases of rachitis, and therefore concluded that the American physicians had confounded the two diseases. More recent observations have established the fact that this bruit has little diagnostic significance. It is heard whenever there is sufficient patency of the anterior fontanelle both in health and disease. It is conducted from the base of the brain through the brain-substance to the membranous covering of the fontanelle. Dr. Wirthgen heard the bruit in 22 of 52 infants, of whom all except 4 were in good health. I have auscultated the anterior fontanelle in 29 infants who were, with two exceptions, between the ages of three and thirty months. All were well or affected merely with trivial ailments which did not disturb the cerebral circulation. In most of them a murmur could be distinctly heard synchronous with the respiratory act, and in 15 of the 29 cases no other sound could be detected, while in the remaining 14 a bruit could be detected synchronous with the pulse.

As might be expected, craniotabes gives rise to symptoms quite distinct from those of the general rachitic disease. It usually occurs during the first year of infancy, and most frequently prior to the tenth month. The brain at this age is soft and yielding, since it contains a large percentage of water. Unless handled with care at an autopsy, it is readily lacerated, and moderate pressure upon it is seen to disturb and move it a considerable distance from the point of contact. It will assist to a proper understanding of the symptoms referable to the cerebro-spinal system to which the rachitic are liable, to recall to mind the fact, well known to surgeons, that slight depression of even a small portion of the skull is likely to produce grave consequences. It is not surpris-

ing, therefore, that craniotabes, when there is a space of considerable size in the cranial arch destitute of bone, is attended by symptoms due to the mechanical effect of external pressure whenever a substance less yielding than the brain comes in contact with the unprotected part.

Every rachitic child is fretful, but one with craniotabes is especially so if the open spaces, in which the lime salts are lacking or constitute a thin and yielding layer, are of considerable size. If the child lie upon the pillow in the position that is most natural for it, the unprotected portion of the brain may be so pressed upon by the weight of the head that it is uncomfortable and restless. It does not have quiet sleep because the cerebral circulation and functions are disturbed because of the fact that the cranial arch no longer protects the brain from undue pressure. Carefully placed in an apparently comfortable position, it awakens often and frets until it is taken in the nurse's arms. Sometimes it instinctively seeks a position on the edge of the pillow, with its face downward, and it becomes more quiet when resting over the nurse's shoulder with no pressure or support upon the cranial arch.

But if fretfulness, disturbed sleep, and the necessity of closer attention on the part of mother and nurse were the only ill effects of craniotabes, it would possess much less pathological significance than pertains to it. Pressure upon so delicate and important an organ as the brain involves risks and produces serious symptoms in proportion to its degree. Even a slight injury of the skull which causes depression, though it may be of trifling amount, will cause serious forms of nervous disorder. Rachitic craniotabes sustains a causal relation in not a few instances to one of the most dangerous of the neuroses—to wit, *laryngismus stridulus*, or spasm of the glottis. Pressure on the cardiac and vaso-motor centres of the medulla in the rachitic infant, in whom reflex excitability is exaggerated, causes contraction of the muscles that close the glottis. It is certain that a large proportion of those who suffer from laryngismus stridulus are rachitic, so that it is more common and severe where rachitis is prevalent, as in England, than where it is rare, as in the rural districts of America. It is not often the cause of death in America, and the fatal cases that do occur are, I think, nearly always in the cities, whereas in parts of Europe, where rachitis is much more common than with us, it is said to cause not a few deaths.

Certain infants when in a state of excitement have what are termed "holding-breath spells." The face is flushed and breathing ceases for some seconds, after which respiration returns and is normal. The attacks are unimportant, but they appear to be the same in nature with the more severe and dangerous seizures of laryngismus stridulus. They have no pathological significance, excepting that they show the same neuropathic state as that in laryngismus, and that they may be precursors of it.

Laryngismus stridulus, or glottic spasm, is usually preceded by more or less impairment of the general health and often by fretfulness, which is characteristic of the rachitic state; but the attack occurs suddenly, without premonition, and is of short duration. It begins with an arrest of respiration, a true apnoea, as if from paralysis of the respiratory centre in the medulla; the lips may be livid, a pallor spreads over the face; sometimes more or less rigidity of the limbs occurs, with carpo-pedal contractions. After a few seconds, a quarter or half minute, a long and deep but difficult inspiration through the narrow chink of the glottis follows, accompanied in many patients by a whistling or crowing sound, and the attack ends with perhaps a momentary appearance of bewilderment or dread on the child's face. Laryngismus stridulus, like eclampsia, does not have a uniform causation. In certain cases it is a reflex phe-

nomenon due to an irritant in some part of the system, as in the intestines, but many observations establish the fact that rachitis is probably its most common cause. A large proportion of the infants affected with it exhibit unmistakable rachitic signs; and it has been held that the exposed state of the brain in craniotabes affords explanation of the symptom. But from observations which I have made and from those of other observers, like Senator, it is certain that laryngismus stridulus is common in the rachitic who do not have craniotabes, so that there must be a causal relation in rachitis to spasm of the glottis independently of the cranial softening.

Distinguished British observers, as Gee and Jenner, have noticed the fact that rachitic infants are especially liable to *eclampsia*. The immediate or exciting cause seems to be in many cases the severe catarrh of the respiratory and digestive systems to which rachitic infants are especially liable. Indigestion, flatulence, and fermentative diarrhoea, common disorders of the rachitic, are perhaps, in some instances, the exciting causes of the eclampsia. Similar remarks may be made in reference to tetany, which, although it occurs in the adult and is comparatively rare, appears to be more frequent in rachitic than in other children.

Those physicians who attend in institutions in which children coming from tenement-houses are treated in a large city like New York have noticed the fact that the various tissues of the body, besides those that are conspicuously affected in rachitis, are more liable to inflammatory diseases than are the same tissues in those who have sound constitutions. The frequency of the different forms of dermatitis, of nasal, post-nasal, faucial, and bronchial catarrhs, and of gastro-intestinal maladies, we must attribute to the fact that rachitis diminishes the resisting power to noxious agents in the various soft tissues, and renders them more liable to disease.

If the deformity in the thoracic wall—to wit, the lateral depression of the ribs and anterior projection of the sternum—be great, we would naturally expect that the two important organs underneath, the heart and lungs, would receive some detriment. Upon the surface of the heart, at the point where it supports the softened ribs, a white patch is often found, due to thickening of the pericardium and proliferation of the endothelial cells, just as thickening of the skin in the palm of the hand occurs from friction and pressure upon that part. It is probable that in ordinary cases this pressure does not seriously impair the function of the heart, but it may increase the weakness of its movements in supervening asthenic diseases, which may occur during the rachitic period. The injury sustained by the lungs is greater and more apparent. If the lateral depression of the ribs be considerable, full inflation of the lungs does not occur in those parts where the depression is greatest. The semi-collapse of certain lobules is likely to occur, and even complete collapse of the distant thin edges of the lungs. The stress of respiration falls unequally upon different parts of the lung. The anterior portion, which ascends with the sternum as that is propelled forward, is more fully dilated than the lateral and posterior parts, and it may in consequence become emphysematous. If in this state of the thorax and lungs severe bronchitis or broncho-pneumonia occurs, the muco-pus, being expectorated with difficulty, clogs the tubes, produces dyspnoea, and imperils the safety of the child. Even in comparatively mild forms of inflammation the result may be unfavorable, owing to the lack of full expansion in the lateral and depending portions of the lung—a condition required to expel the mucus. Severe bronchitis and broncho-pneumonia are the causes of death in not a few cases of rickets attended by marked deformity of the thorax.

RACHITIC PARALYSIS.—In not a few instances in the course of rachitis the use of the limbs is greatly impaired, so as to resemble paralysis, and be designated by this name, though the term “paralysis” is probably a misnomer. Cases like the following, related by Dr. H. W. Berg in the *New York Medical Record*, which closely resemble paralysis, occasionally occur: J. S—, aged two years and eight months, was admitted into the Orthopædic Dispensary Sept. 23, 1885. The parents stated that the child had never walked or stood alone. The legs were wasted, apparently from disease; the patellar reflex was good; there seemed to be some rigidity of the muscles about the knee; and the patient was admitted with the diagnosis of “spastic paralysis.” A closer examination disclosed the fact that the disease was one of typical rachitis, and by the use of the proper diet, with iron and phosphorus, the patient was able to walk in November, and in a few months was entirely cured. The *British Medical Journal*, Jan. 4, 1890, contains the account of a case of rickets discussed by the Edinburgh Medical Society, Dec. 4, 1889. The patient, a boy of three years, had the waddling gait and straddling pose of pseudo-hypertrophic paralysis. The rachitic nature of the malady was made apparent by the symptoms of the case and its history. I have recently in private practice observed two similar cases of pseudo-paralysis of the lower extremities from the same cause.

ACUTE RICKETS.—Occasionally rachitis occurs with the sudden development of severe symptoms, so that the term “acute” is applied to it. Dr. Fürst relates such a case in the *Jahrb. für Kinderh.*, Band xviii. p. 192: The patient, aged two years and one month, had been largely fed upon starchy food, and at six months had dyspeptic symptoms and sweating. Dentition began in the thirteenth month, and ability to walk several months later. Spasmodic croup and swelling of the epiphyses appeared at this time. At the above-mentioned age the child suddenly fell ill with acute febrile symptoms. It had an open anterior fontanelle, craniotabes, and a rachitic chest; upper extremities free from pain and not swollen. The left femur and both tibiæ showed diffuse cylindrical swelling. The appearance and feel of the limbs were suggestive of diffuse cellular infiltration proceeding from the periosteum in an attack of osteo-myelitis. The skin covering the limb was tightly drawn and of a reddish hue. In a few days the right forearm was affected, and soon after the right arm and left forearm, and the parts first attacked began to improve. In four weeks the fever and pain had abated, but swelling of the epiphyses and deformities of various bones continued. Cases like the above establish the fact that although rachitis is ordinarily a chronic disease, insidious in its commencement, gradual and progressive in its development, occupying months, there is an acute form which is attended by more marked febrile movement and tenderness than occurs in the usual type, and in which the articular swelling appears more quickly. (See p. 350.)

Treatment.—**HYGIENE.**—We recall the recent statement of Prof. Hensch of Berlin that the spread of rachitis has been enormous in the cities of Central and Northern Europe. The poor of these cities, among whom this disease largely prevails, are emigrating in large numbers to the United States, but, as I have observed in the asylums and dispensaries of New York, the severest forms of imported rachitis come from Southern Europe (Italy). Evidently, as long as the influx of this class of foreigners continues, and the present insanitary conditions exist in our cities causing rachitis in the native born, this will continue an important disease, impairing the health and vigor of coming generations. It is evident from the nature of rachitis that success in preventing it and in curing those who unfortunately exhibit its characteristic signs

requires beyond anything else the employment of proper hygienic measures. The details of the hygienic requirements may seem prolix and tedious, but we cannot expect any marked diminution of rachitis until they are better known and heeded by the masses.

The fact that inheritance is one of the recognized causes of rickets renders it very important that the parents be in good health. The mother especially should avoid all agencies or influences which impair the general health during the procreative period. She should, so far as possible, encourage good appetite, take plain, easily-digested, and nutritious food, and lead a quiet, regular life, with sufficient out-door exercise to promote, so far as practicable, a state of perfect health. Country residence, with quiet exercise in the open air, a diet consisting of fresh vegetables, meats, fresh and abundant milk, early retirement to bed and sufficient sleep, are much more conducive to the health of the mother and her child than are the excitement and irregularities of city life.

We have seen that there is sufficient clinical and experimental evidence that the common and predominating factor in causing rachitis is the use of a faulty diet, but general insanitary conditions are also potent agents. The foul air and noxious effluvia of the crowded tenement-house, so conducive to disease and fatal to infants in New York, should, if possible, be avoided. Even if poverty compels a residence in the small and dark apartments of a tenement-house, crowded by families, many of them entirely neglectful of sanitary measures, yet parents solicitous for the welfare of their children can do much to diminish the insanitary influences which surround them. Out-door air is everywhere available, and every child after the age of two or three months, unless suffering from acute disease, should in ordinary weather be in the open air one or more hours each day, as a means of improving its digestion and of producing a more vigorous state of the system. Any mother or nurse capable of the care of a child should be able to employ such measures as will prevent its taking cold while in the open air.

The room occupied by a child, whether rachitic or not, should be at a uniform temperature of about 70° to 73° F., and it should receive the sunlight or the full daylight, which is often excluded by faulty construction. The undergarments worn during infancy and childhood should be of wool, thin and light during the summer, thicker and heavier in the winter. No intelligent mother need be told of the need of personal cleanliness of her child as a means of promoting its health as well as comfort. This is a hygienic measure, and we need not repeat that the more complete the sanitary conditions the less the liability to contract rachitis or any disease dependent on cachexia. Bathing of children should always be before the fire or in a warm room. The bath for an infant under the age of six months should be at about 90° . As the age increases the temperature of the bath should be gradually reduced to 80° in the second year, to 75° in the third year, and to 70° subsequently. The bath should be short, only long enough to ensure cleanliness. For weakly infants it is sometimes best to dispense with the general bath, and employ the sponge instead. I see no advantage in the use of saline or medicated baths. After the bath the extremities should be warm, and to ensure a better peripheral circulation friction of the surface by warm flannel or otherwise, or the application of warmth to the limbs, is often useful. The extremities of a child should always be warm, for the normal warmth of the surface not only promotes nutrition of superficial parts, but it tends to prevent internal congestions and inflammations, to which the rachitic are especially liable. A child that habitually has cool extremities cannot be at the maximum of health, and this is often the state of the poorly-fed and poorly-clad children of the tenement-houses. The

measures to promote their normal circulation and warmth, such as exercise as far as practicable, artificial heat, exclusion of cold by woollen garments, friction of the limbs, either dry or by the use of mildly stimulating lotions, should be employed. But while the hygienic measures which we have detailed are important as means of invigorating the system and rendering it less liable to rachitis as well as other cachectic diseases, we repeat that the most common and potent cause of the malady which we are considering is a faulty diet, so that in the endeavor to prevent and to cure rachitis special attention must be given to the feeding.

Clinical experience abundantly demonstrates the fact that in order to promote healthy nutrition the food of the infant should be breast-milk until the age of ten or twelve months; and subsequently, until childhood is well advanced, its food should consist largely of cow's milk, properly preserved and prepared.

We need not state that human milk varies in its composition according to the health, diet, mode of life, and temperament of the individual who furnishes it. Many mothers possess the requisite moral traits to be good wet-nurses, and do all in their power for the welfare of their infants, but have an inadequate lacteal secretion. Many mothers, not only in the tenement-houses, but in the well-to-do class, are unable to furnish sufficient breast-milk, and their infants, unless they receive supplementary food, suffer from malnutrition and are liable to become rachitic. I have seen during the last year infants wet-nursed by their mothers, fretful, wasted, and at the verge of starvation, applied every half hour to the breast during the hours of wakefulness. Mothers, deprived of the needed sleep and sacrificing their own health in the constant endeavor to provide for the wants of their infants, usually have insufficient milk, as in the cases alluded to. Under such circumstances a medicine designated *nutrolactis*, which consists largely of the *Galega officinalis*, has been employed in the New York Infant Asylum with apparent benefit as a stimulator of the lacteal secretion. But if suckling by the mother continue inadequate and her infant be under the age of six months, a wet-nurse should be employed. If this be impossible, supplementary feeding will be needed.

In normal and sufficient wet-nursing the infant should go to the breast at regular intervals of about two hours, but at longer intervals at night (ten times in twenty-four hours). It should obtain what nutriment it requires in ten or fifteen minutes, after which it falls into a quiet sleep. This allows the mother time and opportunity to rest and recuperate between the nursings, so that she furnishes milk more abundant and of better quality than when she is worried and anxious and deprived of needed sleep. The subject is so important that we may be allowed to repeat what we have elsewhere stated: An infant that draws the breast at short intervals of two hours obtains not only more milk, but richer milk, than when the intervals are longer.

There is no more important, and frequently no more perplexing, duty of the physician than to direct the alimentation of infants. Many mothers express the determination to wean for trivial reasons, and are found to be giving one of the commercial foods without consulting the physician. On the other hand, many mothers seriously declare that their babies are ravenous nursers, and that their breasts furnish an abundance of milk, when only a few thin drops can be obtained by the breast-pump, and the appearance of the nurslings plainly indicates innutrition and progressive emaciation. In such cases additional nutriment is of course required.

The practice, which is too common, of early weaning with insufficient reason and without consulting the physician, is very mischievous. Acute and transient ailments of the mother may cause some diminution in her milk, but

usually her health is not so injured by a short sickness that she is incapacitated for wet-nursing; of course the continued loss of appetite, with progressive debility and anæmia, may be such that prompt weaning is imperatively required.

If it be impossible to wet-nurse the infant, or if it have reached the age of ten or twelve months, at which time weaning is proper, it will be necessary to determine what food shall be given. In New York City—and the same is probably true in other cities—the infant should not be weaned in the hot months, since the change of diet from the natural to the artificial at this time is very likely to cause that fatal disease, the summer diarrhœa. The infant should be first removed to the country before weaning, or, if removal be impossible, weaning should be postponed until after the heated term, even if it be at the age of fifteen or sixteen months. But with a large proportion of infants after the age of six months the mother's milk is not sufficient, and it is necessary to supplement the wet-nursing by the use of other foods.

Notwithstanding the many commercial foods designed for infant feeding, I have every year been more and more convinced that cow's milk, properly prepared, furnishes the best substitute for human milk, and should be used to make up the deficiency when the latter is insufficient, and be the main food or the basis of the food employed after weaning. I have observed the occurrence of rachitis in children whose diet consisted chiefly of certain proprietary foods; and, in looking over the composition of these foods, one of the chief causes of this result appears to be the small amount of fat which they contain. Thus, according to Prof. Leeds's analyses, Mellin's Food contains only 0.15 part in 144.74, and Nestlé's Food only 1.91 parts in 139.69, whereas human milk contains 3.90 per cent. of fat, and cow's milk 3.66 per cent. of fat. Especially in the selection of food designed to prevent or cure rachitis our choice should fall on cow's milk next to human milk. But cow's milk contains five times more casein than human milk, and is slightly acid, whereas the latter is always alkaline. In the country, cow's milk obtained fresh and with proper attention to cleanliness in its manipulation may not require sterilization by heat. But that received and used in the city, exposed more or less to an atmosphere containing numerous microbes, it is well to sterilize by steaming for a period not exceeding twenty-five minutes. For infants with feeble digestion, who are suffering from innutrition, digestion of cow's milk can be promoted by peptonizing by the peptogenic powder of Fairchild in the manner well known to the profession. Inasmuch as observations relating to the causation of rachitis, which we have quoted elsewhere, show that deficiency of fat in the food is a common cause, I recommend, especially if any rachitic symptoms appear, the use of the upper half or third of the can or bottle of milk, since this contains a large percentage of cream.

A properly-prepared farinaceous substance, mixed with milk, not only has nutritive properties, but also, by mechanically separating the particles of casein, tends to prevent the formation of curds in the stomach. But as young infants digest starch with difficulty, a flour, as barley, wheat, or oatmeal, in which the starch is to a great extent converted into dextrin, or, better, into glucose, may be advantageously added to the milk, especially for infants over the age of six months. The conversion of starch into dextrin may be effected by a high heat, and into glucose by the action of diastase. If a heaped teaspoonful of barley flour be boiled in twenty-five teaspoonfuls of water, and when it is lukewarm ten or fifteen drops of diastase (Forbes) be added to it, the gruel in a few minutes becomes much thinner from the digestion of starch, and it is a useful adjunct to the milk employed in the nursery, especially for infants over the age of six months.

But while healthy development in infancy and childhood requires a careful choice of food suitable for the stage of growth and development, the frequency of the feeding and the amount of food given are also matters of importance. There can be no doubt that many infants are under-fed, some even to starvation, and some infants are over-fed. MM. Vernois and Becquerel, in a careful examination of 89 infants wet-nursed by mothers apparently in good health, ascertained that 15 were insufficiently nourished. Did space permit I might relate instances in which infants were applied to the breast even more frequently than the prescribed rules allow by affectionate and devoted mothers or by wet-nurses supposed to have sufficient milk, and yet they continued to lose flesh and strength, were almost constantly fretful, and were finally reduced to a precarious state by insufficient nutriment. On the other hand, overfeeding sometimes occurs to the detriment of the child. A half century has elapsed since the most distinguished New England physician of his day, Dr. James Jackson, called the attention of the profession to the frequent, green, and unhealthy stools, showing imperfect digestion occurring in children from over-feeding. Among the cachexiæ developed from abnormal digestion and malnutrition we recognize rachitis as one of the most frequent.

A few years ago Drs. Chadbourne, Parker, and myself made observations in the New York Infant Asylum and New York Foundling Asylum in order to determine how much food children require at different ages. Those selected for observation were well nourished, and they were accurately weighed before and after each nursing or feeding. Eleven infants under the age of three weeks, who took the breast, with three exceptions, twelve times in the twenty-four hours, were found to take on the average 12.55 ounces of the breast-milk in the day and night. Therefore, according to these statistics, infants under the age of three weeks, nourished at the breast and suckled twelve times in the twenty-four hours, require only one ounce, or not more than one ounce and one drachm, at each nursing; and the very small size of the stomach at this age shows that it cannot receive much more than this without distention. After the third week the amount of food required for healthy nutrition gradually increases.

Children, like adults, in good health and well nourished, do not all require or take the same amount of food. Some need more food than others, but the following table indicates, I think, nearly the quantity required during the first twelve months of infancy, either of breast-milk or of food prepared so as to resemble as closely as possible breast-milk in consistence and nutritive properties. It will be observed that this table resembles closely that prepared by Prof. Rotch of the Harvard Medical School, and published in his instructive paper on infant feeding in the *Cyclopædia of the Diseases of Children*:

Quantity of Food required in the First Year of Infancy.

At each Feeding.		Number of Daily Feedings.	Total Daily Amount.
During the first week	1 oz.	10	10 oz.
At the third week	1½ oz.	10	15 oz.
At the sixth "	2 oz.	8	16 oz.
At the third month	3 oz.	8	24 oz.
At the fourth "	4 oz.	7	28 oz.
At the sixth "	6 oz.	6	36 oz.
At the tenth to twelfth month	8 oz.	5	40 oz.

The daily average of food for each child in an aggregate of twenty-eight healthy children between the ages of two and three years was as follows: Bread, 7.5 oz. avoird.; butter, .98 oz.; meat (beef), 4.6 oz.; potatoes, 3.9 oz.; milk, 32.6 fl. oz. The daily average for each child in an aggregate of twelve children between the

ages of three and six years was as follows: Milk, 48.6 fl. oz.; beef, 12.1 oz. avoird.; rice, 13.0 oz.; bread, 10.3 oz.; butter, 1.08 oz. The daily average for each child in an aggregate of twenty-four children between the ages of four and ten years: Roast beef, 12.46 oz.; bread, 10.23 oz.; potatoes, 10.03 oz.; butter, .99 oz.; milk, 38.5 fl. oz.

The prevention and the cure of rachitis require strict enforcement of the details of hygiene. Hence the above facts relating to the mode of life and diet of children should be observed in order to prevent cachexia and promote a healthy growth.

MEDICINAL TREATMENT.—Medicines which aid the digestion and assimilation of properly-selected foods are sometimes useful. Irritability of the stomach, imperfectly digested stools, flatulence, colicky pains, etc. indicate faulty digestion, which may be improved by pepsin given with each feeding. Tonic remedies designed to improve the appetite and digestion, of a kind suitable for the age and condition of the patient, are often useful. In anæmia one of the readily-assimilated preparations of iron should be given. The complications which are so common require special management. The laryngismus stridulus, eclampsia, and tetany should be promptly treated.

The bronchial catarrh to which rachitic infants are liable may be best treated by remedies like the following:

℞. Ammonii chloridi ʒj.
Syr. Tolutan. f ʒij.—M.

Sig. Dose fifteen drops every hour or two hours for an infant of six to ten months.

℞. Ammonii chloridi
Ferri et ammonii citratis āā ʒss.
Syrupi f ʒj.
Aquæ f ʒij.—M.

Sig. Give one teaspoonful every two to four hours to a child of one year.

Some of the rachitic cases with protracted bronchial catarrh, especially those which also exhibit scrofulous symptoms, may be most relieved by the syrup of the iodide of iron and cod-liver oil administered three times daily, with the inhalation of moist air containing turpentine vapor.

In the protracted intestinal catarrh of rachitic infants I have observed the best results, so far as medicine is concerned, from the following prescription:

℞. Subnitrate of bismuth ʒij—iij.
Essence of pepsin (Fairchild's) f ʒj.
Distilled water f ʒij.—M.

Sig. Shake bottle; give half to one teaspoonful, according to the age, every two hours.

But a remedy is needed which will act promptly in the cure of rachitis so as to prevent the evil consequences which its continuance is sure to produce. It is the opinion of many of the best clinical observers who have had ample experience that this has been discovered in the daily use of minute doses of phosphorus.

Wegner fed young and growing animals (rabbits and fowls) for months with small, non-poisonous, and easily assimilated doses of phosphorus, with the result, he believes, of expediting ossification and producing firmer bone.

He states that under the influence of phosphorus the large marrow spaces diminish, by the formation of true bone, to the size of the Haversian canals in normal bone. According to Wegner, the administration of finely-divided, non-poisonous doses of phosphorus for a prolonged period to older fowls produced to a considerable extent the conversion of cancellous into compact bone of normal chemical composition. Kassowitz has recently promulgated his views at some length on the pathology and treatment of rachitis. He states that the lime salts are not needed, since the ordinary food contains sufficient lime; nor should the farinaceous foods be restricted. He adds that phosphorus in small doses restricts the formation of vessels in the growing bones of small animals. Hence it is useful as a means of overcoming the hyperæmia. Kassowitz administers about $\frac{1}{133}$ of a grain in a teaspoonful of cod-liver oil, the dose, of course, varying according to the age of the infant. The distinguished pædiatrist of Vienna, Dr. Widerhofer, says of this remedy that its employment "impresses him with the belief that it is not without benefit in the second year of life and upward." He thinks that it may be useful in the hardening of long bones, but he has not been able to obtain good results in craniotabes. Stärker gives an analysis of 23 rachitic cases treated by Prof. Thomas of Freiberg in his clinic. He used the following formula:

R_y. Phosphori 1 centigramme (about $\frac{1}{7}$ grain).
 Ol. morrhue 100 grammes (about 3 ounces).—M.

A coffee-spoonful was administered twice daily, but variations in the dose according to the age are not stated in the report, the patients being between the ages of a few months and four years. Improvement occurred in the general condition in 18 cases; in the cranial development in 15 cases; in dentition in 14 cases; in the shapes of the epiphyses in 21 cases; in locomotion in 17 cases; but strict attention was bestowed upon the hygiene, and especially upon the diet. Soltmann states that good results occurred from the use of phosphorus in 70 cases which he had under observation, and in no instance were unfavorable results noticed. W. Meyer obtained similar results in 42 cases. He regards phosphorus as a specific for rachitis. When properly given it always, says he, produces positive results. Petersen has treated 200 cases with phosphorus, and regards it as a specific. Sigel concludes, from the observation of 40 cases in private practice, that constitutional treatment is of the greatest importance, but instead of the administration of iron, lime, etc., phosphorus should be prescribed. Unruh also made many observations in the treatment of rachitic cases by phosphorus in the Dresden Hospital in 1885 and 1886, and considers it more efficacious than other remedies.

Toplitz of Breslau treated 518 cases with phosphorus combined with cod-liver oil. No ill effects were observed, and in all the cases improvement occurred in the general condition. Of 208 cases of craniotabes, 176 were cured in eight weeks. In 58 cases of laryngismus stridulus the attacks ceased in eight to fourteen days, after having continued for months under other forms of treatment. Dentition was also promoted.

In America, Dr. A. Jacobi, who has had a large clinical experience, also highly recommends phosphorus in the treatment of rachitis. The dose should be small, even minute, not more than $\frac{1}{200}$ to $\frac{1}{100}$ of a grain, according to the age, three times daily.

As regards my own observations, I am not able to express a positive opinion as to the value of the phosphorus treatment, for reasons which I think also apply to many of the cases embraced in the favorable statistics of the dis-

tinguished observers mentioned above—to wit, the simultaneous use of cod-liver oil and improvement in the diet and general hygiene.

The following prescriptions may be employed—first, the oleum phosphoratum, made according to the following formula:

℞. Phosphorus 1 part.
Ether 9 parts.
Almond oil 90 “ —M.

One minim contains $\frac{1}{100}$ of a grain of phosphorus.

Or, secondly, the following, known as Thompson's mixture:

℞. Phosphori gr. j.
Alcoholis (absolut.) ℥ cccl.
Spts. menth. piperit. ℥ x.
Glycerini f ʒij.—M.

Sig. Six drops, increased to 10, three times daily, to a child of two to four years. Ten minims contain $\frac{1}{32}$ of a grain, and thirteen minims contain $\frac{1}{100}$ of a grain.

Phosphorus should, I think, be given after the meals, in order to prevent irritation of the stomach.

Dr. H. H. Purdy, physician to the large class of children's diseases in the out-door Department at Bellevue, has preserved statistics of the treatment of rachitis during the last year. The cases which furnish the statistics numbered about 80, and he gives a résumé of the results of treatment as follows: “Some were given cod-liver oil alone, some, cod-liver oil with phosphorus, and others, phosphorus alone, and of course all the mothers were given instruction in feeding and hygiene. Those infants that received only phosphorus were the slowest to improve. Indeed, in several cases this method of treatment was abandoned because of the absence of the signs of improvement. The group treated with cod-liver oil did the best. In fact, all of the infants that could tolerate the oil derived much benefit from it. The group that were given cod-liver oil with phosphorus did very well, but seemingly no better than those that were given only cod-liver oil. The preparation that seems to be the most beneficial is one that is used at the Church Hospital and Dispensary. It is an emulsion of cod-liver oil made with the yolk of eggs. The formula for the emulsion is:

℞. Yolks of ten eggs.
Cod-liver oil Oij.
Syrup of wild cherry Oj.
Sherry wine Oj.—M.

Sig. One or more teaspoonfuls administered three or more times daily.”

In my opinion the treatment by phosphorus is still tentative, notwithstanding its recommendation by so many distinguished physicians; and the old remedies, cod-liver oil and iron, should not be abandoned, although trial may be made of phosphorus at the same time.

Care should be taken to prevent deformities while the bones are soft and yielding. The patient should not be encouraged to stand or use the limbs until they become firmer. He should lie upon a soft and even mattress. Uniform support of body and limbs is requisite in order to prevent curvature. In craniotabes the pillows should be soft, and care should be taken that the yield-

ing parts of the cranium be not unduly pressed upon. Profuse perspiration may be relieved by sponging with vinegar and water. The patient may be bathed in water a little cooler than the body, and rock salt may be added to the bath.

The attacks of laryngismus stridulus, eclampsia, and tetany which so frequently complicate rachitis should be promptly treated by the remedies which are appropriate when they occur under other circumstances. Constipation may be treated by enemata of glycerin and water if not relieved by change of diet.

The surgical treatment of rachitic deformities is sometimes important, but Prof. Ogston of the University of Aberdeen and other surgeons who have given special attention to this subject state that in young patients these deformities frequently diminish during growth, so as to cause little inconvenience in adult life. The measures employed by surgeons in order to cure or minimize the deformities are fully set forth in surgical treatises.

[**Acute Rickets.**—It is now generally accepted by American and English observers, that the condition sometimes described as “acute rickets” is in reality scorbutic in nature. This is certainly true of the cases reported by Möller, Bohn, Förster, and Senator. The case of Fürst, quoted by Dr. Smith on page 342, which showed diffuse cylindrical swelling of both tibiæ and of the left femur, is certainly very suggestive of scorbutus, despite the fact that the statement is distinctly made, “no scorbutus, no stomatitis.” In this case it can only be said that “acute rachitis” is “not proven.”—ED.]

RHEUMATISM.

BY J. M. DACOSTA, M. D., LL.D.,

PHILADELPHIA.

I. ACUTE RHEUMATISM.

ACUTE RHEUMATISM, or rheumatic fever, is a specific febrile malady characterized by inflammation of fibrous tissues, particularly those surrounding the joints, of which many are apt to become affected simultaneously or in succession. There is also in rheumatism a strong tendency for the serous membranes, especially those of the heart, to become involved, and in children we frequently find these bearing the brunt of the disease while the articular affection is very slight.

Etiology.—The cause of rheumatism is the accumulation of some poisonous matter in the blood which irritates specially the fibrous and serous tissues. The most commonly held opinion is that this poison is lactic acid, though the evidence is far from conclusive. The lactic acid may be the result merely of the morbid process, not the cause. Though sought for, specific micrococci have not been demonstrated, nor has the origin of acute rheumatism in disorder of the nervous system been proved.

But, whatever the remote cause, it is certain that chilling of the surface is in the majority of instances the immediate cause producing the attack. A history of exposure to cold and damp can be almost always obtained. In instances, on the whole infrequent, the poison of scarlet fever produces pain, swelling of the joints, and even cardiac symptoms indistinguishable from acute rheumatism.

The most potent predisposing cause of acute rheumatism in the young is hereditary tendency. Out of 492 cases Cheadle found a distinct history of its occurrence in near blood relations in 173. The strong hereditary tendency is also illustrated by the experience of Steiner: of 12 children of a mother who had suffered from acute rheumatism and heart complication, 11 had the disease before they were twenty years of age. Besides the complaint running in rheumatic families, I have noticed that the children of gouty parents develop rheumatism in greater proportion than found in those free from gouty taint. With reference to sex, unlike what happens in adult life, acute rheumatism is more common in girls than in boys. It is not often seen before six years of age. Yet August Seibert met with rheumatism in 13 children under one year of age, and cases of its occurrence in very young infants are recorded by Henoeh, Senator, and Koplik. A case of acute rheumatism in an infant eleven days old is reported by Guthrie, and two remarkable instances of its manifesting itself soon after birth are mentioned by Jaccoud: one showed itself three days, and another twelve hours after birth, the mothers at the time being ill with acute rheumatism. I have myself met with a case of acute rheumatism under two years of age. This happened in a girl the daughter of a

highly gouty father. She has now grown to womanhood, having had three severe attacks of rheumatic fever, but without the heart becoming affected.

Morbid Anatomy.—The joints show an injected synovial membrane, and there is effusion of fluid into them and into the surrounding tissues; the fluid contains blood-cells and sometimes leucocytes. Minute hæmorrhages into the membrane are not uncommon; the cartilages are swollen, but it is very rare for them to suppurate or to ulcerate. Near the affected joints and tendons fibrous nodules similar to those found on the valves of the heart are met with, and the parts around the joints, as Hænoch has called attention to, may be infiltrated with inflammatory exudation that even becomes as hard as bone. Nodules growing from the bone, a nodular periostitis, have been described by Angel Money. In the heart inflammatory lesions are usual, both in endocardium and in pericardium. The pericarditis in the acute rheumatism of childhood, Cheadle has pointed out, frequently extends to the anterior mediastinum, the connective tissue of which becomes extensively thickened. The extent of pericardial effusion is not generally great, but there is much plastic exudation in the membrane. Fibrinous coagula are found in the heart and great vessels. Pleurisy with or without effusion is often seen.

Symptoms.—The symptoms of acute rheumatism in childhood are the same as those of adult life: redness and swelling of the larger joints, pain, fever, perspiration, heart involvement. But these symptoms do not occur in the same degree. The *joint affection* is apt to be slight—certainly the swelling and redness are—while stiffness and tenderness may be marked. The joints become successively involved, but in children it is not uncommon to find the rheumatic inflammation limited to a very few joints, such as the ankles or the wrists. Even there it may be pain and tenderness rather than swelling that arrests attention. It is on account of the slight joint affection that acute rheumatism in children is often overlooked, and the pain and tenderness are attributed to a fall or a sprain until the damaged heart tells the story.

The *fever* is not high or long-continued; it is seldom above 102° F. Of those terrible cases with high temperature—temperature reaching from 107° to 110°—of which I have met with many in adults, I have never seen an instance in childhood. Fagge observed in 14 cases of the dreaded complication not one less than eighteen years of age; Wilson Fox, in 22 cases none less than seventeen years; Barlow records a fatal case in a girl of thirteen. Hyperpyrexia is certainly most unusual; and so are the cases with delirium and other signs of cerebral disorder, and the cases with typhoid symptoms, whether associated with high temperature or not. Where the febrile rise is high and protracted there is apt to be delirium, and the morbid signs generally depend upon a heart affection, especially pericarditis. The tongue is not so coated as it is in adults; the urine is high-colored, dense, with an excess of lithates. From among the usual symptoms of rheumatic fever we miss in children the profuse acid sweats. The skin is moist, but not bathed in perspiration.

The *heart symptoms* of the rheumatic fever of childhood occur very commonly; indeed, in children endocarditis and pericarditis are more usual attendants on acute rheumatism than in adults. Endocarditis shows itself by increased restlessness, hurried breathing, dry cough, uneasiness or pain in the cardiac region, a rise in temperature or at least a sustained fever temperature, and the development of a murmur, which is generally at or near the apex and systolic. This mitral murmur is followed by an accentuated second sound, or its reduplication, at the apex; in rarer instances in place of a mitral an aortic murmur is present; in yet rarer instances there is a diastolic aortic murmur, or a diastolic or a presystolic mitral murmur. The impulse is some-

what increased in force, slightly in extent, but the percussion dulness, difficult to ascertain in a child, is not distinctly altered. The pulse becomes more tense, and its beats are not equal. As the case advances, impaired pulmonary resonance and fine râles indicative of congestion may be noticed, and restlessness and anxiety and irregularity of the circulation augment. Where ulcerative endocarditis takes place, recurring chills like those of malarial fever, followed by high temperature and profuse sweats, are apt to occur. And both in this form and in the simple form of endocarditis masses of fibrin may be washed from the vegetations into the vessels of the brain or elsewhere, and cerebral embolism or embolic pneumonia or other kinds of embolism thus happen.

Besides the marked forms of endocarditis we may encounter only dulness of the first sound, giving it a murmurish character, without decided general symptoms attending the ill-developed cardiac changes. These are instances of mere swelling and slight inflammation, and rarely result in persistent alteration of the valves, as the cases with well-defined murmur commonly do. Then, again, it must be borne in mind that there are many cases in which the general symptoms are so slight that the endocarditis readily escapes detection. Indeed, it is alone the recognition of the changes in the heart-sounds that makes sure of the presence of the malady.

Pericarditis, owing to the greater difficulty of its recognition, is more often overlooked than endocarditis. This is especially the case in very young children, in whom, however, it is not common. It may occur at any stage of rheumatism: sometimes it precedes the joint affection; often it pursues a sub-acute, irregular course, subsiding and breaking out anew as fresh joints become involved. The symptoms are those of endocarditis, but there are greater restlessness and distress, more marked signs of nervous disorder, a tendency to higher temperature, more cardiac pain. The physical signs are the same as in the adult; prominent among them is the friction-sound, followed, when effusion takes place, by increased percussion dulness, by disproportionate distinctness of the sounds at the base as compared with those of the apex, by muffled sounds at the apex, and its upward displacement. It is much more difficult in children than in adults to make out the dulness, or to determine its triangular shape or its existence in the fifth interspace to the right of the sternum; and very often the dulness is of irregular shape, and dependent upon thick layers of plastic pericarditis, indicating its existence by coarse friction and by the sounds of the heart being much the same at the apex and at the base. This form of pericarditis without liquid effusion is, indeed, common in childhood.

So is *pleurisy* as an attendant upon acute rheumatism common, and not only single pleurisy, likely then to be left-sided, but double pleurisy. One of the dangers of left-sided pleurisy is that the inflammation is apt to spread to the pericardium; at all events, whether from contiguity or from simultaneous action of the rheumatic poison, pleurisy and pericarditis are often combined, and both may be of the exudative plastic variety rather than attended with effusion. Still, effusion does happen in rheumatic pleurisy, and may be of slow absorption or become purulent. *Pneumonia* rarely complicates the pleurisy; when it does, it may only reveal itself by rise of temperature, without marked cough or expectoration, and by the physical signs. Cheadle believes that these are different from those of pneumonia in the absence, except in the embolic form of the malady, of fine crepitation.

Chorea bears a very close relation to the rheumatism of childhood. Rheumatic children are very apt to be irritable, nervous, emotional children, and

therefore with nervous systems predisposing to chorea. The chorea associated with acute rheumatism has, in my experience, most generally shown itself toward the end of the attack and when the acute symptoms have disappeared. In the majority of instances there has been pericarditis or endo-pericarditis. Sometimes the choreic movements begin at the height of the malady, or the chorea even precedes the joint affection. It must further, in estimating the relation of chorea to rheumatism, be borne in mind that chorea does not always follow an acute attack, but may come on in those of rheumatic taint, without previous well-defined rheumatic manifestations.

Cutaneous eruptions are often seen in the rheumatism of childhood. The most common form is erythema, which appears on the limbs and the body, and is of the papulated or margined form, or shows itself as urticaria, less often as erythema nodosum; in rare instances it is purpuric and associated with subcutaneous hæmorrhages. Barlow has pointed out that the erythematous rashes may appear simultaneously with pericarditis, or precede this and the articular symptoms.

But more important than these rashes, and much more strictly linked to rheumatism, are the *fibrous nodules*. Of extreme rarity in adults, they are not uncommon in children. They are mainly to be found about the joints, are hard and painless or slightly tender on pressure, of size varying from a pin's head to a cherry, and are chiefly to be ascertained by the touch. They come and go in a few weeks, though they may last for months. It is not unusual to have them appearing in crops, and, though these subcutaneous nodules may project from the surface, the skin over them is not discolored. They are almost constantly associated with endocarditis or with pericarditis, and when abundant and frequently recurring imply a progressive cardiac affection.

Among disorders we frequently meet with in the rheumatism of childhood is *tonsillitis*. It is often antecedent to the rheumatic attack or occurs in its course, and is combined with decided rise of temperature and pain in swallowing. It is not followed by either ulceration or suppuration.

The *anæmia* that attends the rheumatism of childhood is very pronounced, and persists long after the attack. Where successive rheumatic seizures occur it becomes more and more decided, and is often associated with marked irritability of the nervous system and emotional disturbance. In its persistence it may become a factor in the mischief wrought by a heart disease and in the development of dropsy.

Diagnosis.—The diagnosis of acute rheumatism in a child is more difficult than in an adult, because the joint affection is often very slight, and may be nothing more than mere stiffening attended with moderate fever, or pain in moving certain muscles and tendons. Under these circumstances we have to lay great stress on the family history, on the character of previous seizures, on the occurrence of attacks of tonsillitis. Signs of endocarditis or pericarditis, or pleurisy, or erythematous rash, or nodules, would be conclusive. In some instances, too, epistaxis, an occasional symptom of the rheumatism of childhood, is very significant; so is chorea. Endocarditis or pericarditis in a doubtful case would be, however, the most certain of proofs.

When the joint affection is distinct, scarlatinal rheumatism is the disease most likely to be confounded with ordinary acute rheumatism. As regards the symptoms I know no difference; heart affections in scarlatinal rheumatism are less common, but they arise. I have sometimes thought the absence of sweating diagnostic, but the acid sweats of rheumatic fever are also often absent in the rheumatism of childhood. Nothing but the antecedent history makes the case absolutely certain. The severe pain and the swelling

of the joints sometimes observed in cerebro-spinal fever may cause this to be mistaken for rheumatism. But the violent headache, the retracted head, the rosy or petechial eruption, the irregular temperature and pulse, are very different from the combination of symptoms noticed in rheumatic fever. In its earlier stages rickets may mislead, on account of the swelling near the joints, the pain, the sweats, the fever. Yet the absence of redness of the joints, the size of the epiphyses, the undisturbed heart, the cachexia, the pale urine, and the fact that the wrist-joints are apt to be the ones first disturbed, or that the swelling shows itself chiefly on the dorsum of the foot and on the back of the hand, are full of significance.

From pyæmia, rare in children, rheumatism differs by the irregular fever of the former, the sweats, the great pain and swelling that are found in only one or in a few joints, and the course of the disease. There is a pyæmic arthritis to which infants are liable, that Townsend has well described, which runs an acute course, is mostly confined to the hip or knees, and in which the effusion speedily becomes purulent. Its occurrence in infants at the breast or when gonorrhœal ophthalmia or vaginitis is present also distinguishes it.

Scurvy may present pain and swelling of the joints; the absence of fever and the condition of the gums tell us that it is not rheumatism. In congenital syphilis the state of the bones near the joint may lead to the thought of rheumatism, but the characteristic eruption, the snuffles, the emaciation, the enlargement of the spleen, the rarity of fever, and the fact that the symptoms arise in early infancy are diagnostic.

The diagnosis of the most dreaded affection in rheumatism, the endocarditis, presents the same points for consideration as it does when it is not of rheumatic nature, and is discussed in another part of the volume. I will only here mention how important it is to remember the anæmic state that rheumatism develops in the young, and not to regard every murmur arising in its course, and especially when it has nearly run its course, as organic and as likely to lead to permanent valve-injury. These soft, systolic blood-murmurs are unconnected with change in valve or in muscular texture, and gradually pass away.

Course and Duration.—The course of acute rheumatism in childhood depends very much upon the complications, especially upon the cardiac lesions. Nor do we find as many frank cases running their course in a definite time; the cases are mostly subacute, with subsidences and fresh outbreaks. On the other hand, in infants there are instances of very rapid progress. Jaccoud's cases in infants soon after birth terminated, one in eight days, the other in little more than two weeks. As a general rule, the rheumatic fever of childhood lasts between two and three weeks. Slight cases, Steiner estimates, get well in from ten to fourteen days. Goodhart's results in ten cases, of which he stated that the longest duration was four days, is not the general experience. It is difficult to be precise in this matter of duration, since much depends upon how early the patient has come under treatment and how well he responds to treatment. Under the salicylates we see the duration often much abridged, in instances particularly of joint affection without internal lesions. Where the heart is affected the case frequently runs on for five or six weeks. Frank relapses are not common. But a succession of subacute attacks in rapid succession, affecting the joints but slightly while adding to the mischief in the heart, is not uncommon.

Prognosis.—This is favorable; few die in the disease. Certainly this is true of the first attack; if the attacks be repeated, there is much more danger during the acute seizure. And the danger, again, depends rather upon the

condition of the heart than upon the mere recurrence of the rheumatic fever. The liability to cardiac disease increases with the number of attacks. Yet this does not always happen. I have mentioned a case in which three severe attacks happened without heart implication; and A. Clark tells of one in a boy of twelve in which eight attacks occurred, the heart remaining sound. Such instances are, however, very exceptional. Age has something to do with the prognosis. Of cases between one and ten years of age, 83 per cent., McPhe-dran calculates, have heart lesions; between ten and twenty, 69 per cent. In 54 fatal cases of rheumatic heart disease Sturges encountered none under two years of age; 42 out of the 54 happened between six and twelve years. Embolism and thrombosis are rare, but very grave.

The chief concern where cardiac affections exist is as regards the amount of mischief that will remain after the acute symptoms have subsided. A murmur indicative of mere roughening of the valve may in the course of a few months disappear. But very often it persists, and gradually, if the lesion have been more than mere roughening of the valve, the signs of hypertrophy with dilatation become manifest. This may not happen from the first attack; but during slight recurring rheumatic seizures—slight at least so far as the joints are concerned—the heart affection is little by little added to; or this is aggravated by a more severe attack, in which a fresh extensive endocarditis occurs. From pericarditis we may have the same consequences as in adults—adherent pericardium with hypertrophy or dilatation; considerable effusions are very rare. Rheumatic pericarditis by itself has a better prognosis, both at the time and in its ultimate consequences, than endocarditis. But with reference to the latter it must be borne in mind that it is mostly associated with some pericarditis, really an endo-pericarditis; for few are the cases where endocarditis of rheumatic origin alone exists. Persistent anæmia after rheumatic endocarditis or pericarditis is always a bad sign. The hypertrophy or dilatation, which under any circumstances happens more rapidly in children than in adults, gains at increased rate. The frequent occurrence of fibrous nodules is a sign of danger, as fresh mischief is apt at the same time to be wrought in the heart. It is then here, as it is throughout in acute rheumatism, the heart, after all, that chiefly determines the prognosis. Chorea is rarely a serious complication. The joint affection mostly passes off completely; rheumatic thickening and ankylosis are very seldom seen in childhood.

Treatment.—The treatment of acute rheumatism in a child is the same as in the adult. The greatest care must be taken to keep the patient at rest and from being chilled, and with this view the child should be kept in bed in a flannel night-dress or between blankets. The diet should be at first chiefly farinaceous, with bread and moderate amounts of milk; later in the disease broths and fish may be allowed. Of medical remedies, the most prominent is salicylic acid or its compounds; among these, salicylate of sodium or of ammonium is well adapted. The dose to a child five years of age is thirty to forty grains in divided doses in twenty-four hours; to a child of ten, sixty to eighty grains. It may be given in syrup of orange, or in simple syrup with spirits of lavender. The salicylates relieve the joint affection and the pain, and their action is rapid; after the third or fourth day the dose may be diminished one-half or more. If no result be seen from them in three or four days, they are not likely to produce any, and some other remedy had better be administered. Nor ought they to be trusted to where heart complications exist. Further experience, indeed, both in children and in adults has only added to my conviction, expressed some years since, that the salicylates neither prevent pericarditis or endocarditis, nor benefit its course after it has set in. Their

chief use is where there is much pain and the joint affection decided; and it is always well in any case to give also alkalies from the start. When the circulation becomes depressed, or buzzing in the ears or giddiness occurs, the salicylates should be at once discontinued. Salicin is by some recommended as less objectionable, in doses of from five to eight grains every third or fourth hour to a child of five, after the salicylates have been administered for a day or two, or even from the beginning.

Under any circumstances, in instances of heart complication or where a heart lesion has existed from a previous attack, the alkalies are vastly preferable remedies. It is, indeed, to decided doses of the alkalies that we must trust. Fifteen to twenty grains of bicarbonate of sodium in simple syrup and mint-water every third or fourth hour to a child eight or ten years of age, or two drachms of the acetate of potassium in divided doses in the twenty-four hours, form the proper average dose. These alkalies should be administered until the urine becomes alkaline or neutral, and then enough be ordered to keep it neutral.

Quinine is very valuable. It may be given in decided doses when the temperature tends to run high, as, however, it is not apt to do in children unless there be endocarditis or pericarditis. In doses of about six grains daily to a child five years of age it is an excellent remedy when the more acute symptoms have subsided, whether the alkaline or the salicylate treatment be the one pursued.

Opium is another remedy of great value. It allays restlessness and pain and procures sleep. In coexisting endocarditis or pericarditis it may be directed in small, continuous doses, and is indispensable. The bromides relieve restlessness and excitability, and are not without influence on the course of the disease. Conjoined to chloral, they give rest at night; and Goodhart lauds the combination of five grains of the bromide of potassium and one or two of chloral as almost a specific for the nightmare of rheumatism in young children.

The treatment of the main internal lesions, the endocarditis and the pericarditis, is discussed in another part of this volume. I will only here speak of my favorable experience in pericarditis with brandy or whiskey in decided quantities, and with opium. The pleurisy is treated as all pleurisies are; the iodides are especially applicable to the plastic form. The salicylate of sodium has been recently highly spoken of in this kind of pleurisy; I have had no experience with its use. In the tonsillitis of rheumatism the salicylates give quick results.

The local treatment of rheumatism consists in wrapping the affected joints in cotton wool, or, where they are very painful, in a flannel bandage saturated with a solution of nitrate of potassium, one to two drachms to the ounce, to which laudanum, twenty drops to the ounce, has been added. For lingering swelling of the joints the rubbing in of iodine, ten to twenty grains to half an ounce of lanolin and half an ounce of belladonna ointment, is well adapted. During convalescence iron is strongly indicated; and there should be then, as always in rheumatic children, the greatest care exerted with reference to warm clothing, to the food being of easily digestible kind, and to the avoidance of exposure to cold and damp as well as to fatigue and over-exertion.

II. MUSCULAR RHEUMATISM.

This is met with in children, as it is in adults, mostly following cold and exposure, especially exposure to draughts, or fatigue. The disorder is generally subacute, and attended with but little constitutional disturbance. The prom-

inent symptom is pain in moving the parts involved. It is very rarely a general disorder, but is limited to particular groups of muscles. We find it in the deltoid; or in the muscles of the loins, as lumbago; or giving rise to stiff neck, as torticollis; or involving the intercostal muscles and restricting the acts of breathing, as pleurodynia; or in the muscles of the head, as cephalodynia. Wherever it is, it has the same characteristics—pain on motion, slight tenderness, little if any fever. Not unfrequently the urine is high-colored and full of urates.

Diagnosis.—In the diagnosis of the affection we have to distinguish it from neuralgia. The stricter limitation of the pain of neuralgia to particular spots, and its passing along special lines of nerve-distribution, the far less influence motion has on it, form, broadly speaking, the traits of distinction. We must also not be misled in considering as muscular rheumatism “growing pains,” or the pains of aching muscles after unusual exercise.

Prognosis.—The prognosis is always favorable. The main object, when the immediate attack has been remedied, is to prevent recurrences.

Treatment.—Rest of the affected muscles, the application of warmth by hot fomentations or the hot-water bag, the use of liniments containing chloral, chloroform, or opium, are all beneficial. Atropine and morphine hypodermatically, so valuable in adults, cannot be so generally employed in children. Diaphoretics are always serviceable; a combination of nitrate of potassium and Dover’s powder is eminently so; and in lingering cases the bromide of ammonium or the iodide of potassium or of ammonium is of distinct benefit. So is the continuous current. Jacobi considers that the best preventive is the habitual use of cold water.

III. CHRONIC RHEUMATISM.

Chronic rheumatism, as we see it in adults, is rare in children; certainly long-continued stiffness of muscles and chronic enlargement of joints are rare. As already pointed out, recurrence of short attacks with stiffness and pain is the form in which the persistency of rheumatism in childhood much more generally shows itself.

The few cases that present the same appearances noticed in the chronic rheumatism of adults may be mistaken for rheumatoid arthritis—a disease which is not unknown in childhood, though it is rarely spoken of. The previous history of the case, the occurrence of rheumatoid arthritis in those of feeble health, the wasting of the muscles, the enlarged, crepitating, or fixed joints with the gradually developing characteristic distortion of the fingers and toes, and the absence of all tendency to cardiac affection, are significant in the distinction.

In the treatment of chronic rheumatism the chief remedies are the iodides, the muriate of ammonium, and arsenic, with great attention to general health and thorough protection by dressing warmly. Using iodine to the affected joints or rubbing them with ammoniated liniments, or, if there be effusion or bony thickenings, small blisters applied from time to time, will give the best results. Good is also done by massage, and by warm baths with carbonate of sodium dissolved in them, or by a recourse to the sulphuretted and alkaline mineral-water springs that have been found to be of real service in the chronic rheumatism of adults.

PART V.

DISEASES OF THE BLOOD.

ANÆMIA, SPLENIC ANÆMIA, LYMPHATIC ANÆMIA, AND LEUKÆMIA.

BY FREDERICK A. PACKARD, M. D.,

PHILADELPHIA.

WHILE in most respects the blood of infants and children resembles that of adults, there are in the blood of the new-born a few variations from the adult standard which require mention.

During the first twelve days of life the blood has a somewhat venous appearance when seen in bulk.

In the new-born child the red blood-corpuscles are of much more unequal size than they are in older children and in adults, the largest of them being larger, and the smallest, smaller. During the first four days of life there are to be found a varying number of nucleated red cells. These soon disappear, although some observers claim that they are to be found up to the second or third year.

Owing, presumably, to the ready solubility of the hæmoglobin in young infants, numerous "shadows," or red blood-cells that have lost their hæmoglobin, are present. The red cells are more easily affected by reagents than is the case in adults, moisture in particular causing them to very readily assume the spherical form. The number of red cells is proportionally larger in the newly-born, the count varying, according to different observers, from 4,300,000 (Bouchut, Dubrisay) up to 7,500,000 (Gundobin) per cubic millimetre. The daily variations in their number are very marked.

There is marked increase in the number of colorless blood-cells in young infants as compared to adults. The subject of the relative number of the different forms has been most carefully studied by Gundobin (*Jahrb. f. Kinderheilk. u. phys. Erziehung*, Bd. xxxv. Hft. 1 and 2, Jan., 1893). According to this author, the relative percentage of lymphocytes in sucklings is three times as great as in adults, while the neutrophiles are relatively twice as small in number. From the seventh to the tenth day is the period at which the relative and absolute numbers attain the proportions maintained in later life.

The amount of hæmoglobin is greater in young infants than in adults. This relative increase is maintained for some weeks, at the end of which time it begins to diminish, until at about the middle of the first year it has reached its lowest point, thereafter slowly increasing to the normal of adult life.

The specific gravity is said to be high immediately after birth (1.066), but it soon sinks to a little below that of adult blood.

PLETHORA.—It is now granted that, while this term may be used as a convenient means of describing certain conditions, it is not accurate, in so far as

it implies an actual increase of the total mass of blood or of its corpuscular elements. The term was employed to indicate a condition formerly supposed to be due to "full-bloodedness," but now known to be a condition wherein the appearance of vascular turgescence is due not to any over-richness in blood, but to local changes in the superficial vessels. That a relative increase above the normal of the number of red blood-corpuscles can exist is true only in conditions where the watery constituents are decreased, as in cholera. To this condition the term "plethora" is manifestly inapplicable, the loss of fluid merely increasing the number of corpuscles in the drop.

ANÆMIA.

ANÆMIA is a condition of the blood due to a decrease in its richness in either corpuscular elements or hæmoglobin, either from primary disease in the blood-making or blood-destroying organs, or, secondarily, from general or local disease that interferes with normal absorption, metabolism, and assimilation, or is productive of abnormal loss of nutritive material from the body.

In the above definition anæmia is spoken of as a condition instead of as a disease, since in the vast majority of instances it is merely a symptom of some well-recognized disease of the whole body or of individual organs. The anæmias produced by morbid processes that are recognizable as distinct diseases are spoken of as secondary, whereas those occurring without apparent cause save disease of the blood-making or blood-destroying organs are spoken of as primary. In the latter class we must still place chlorosis, progressive pernicious anæmia, splenic anæmia, lymphatic anæmia (Hodgkin's disease), and leukæmia.

SECONDARY ANÆMIA.

Etiology.—Our knowledge of the process of blood-formation and blood-destruction is not sufficiently advanced to explain the production of anæmia in all cases in which it occurs. Where actual escape of blood from the blood-vessels takes place, the explanation is, of course, manifest; but it is far from evident in exactly what manner prolonged high temperature, loss of albumin from continued suppuration or Bright's disease, the rheumatic poison, and certain toxic influences produce decrease in the richness of the blood in corpuscles or hæmoglobin. In childhood the chief causes of secondary anæmia, aside from those operative equally in adult life, are due to improper hygiene as to diet, exercise, and ventilation. A frequent cause is mucous disease, which seems to act by preventing the proper digestion, absorption, and assimilation of nutritive material. Improper articles of diet and improperly prepared food may act in practically the same way; that is, by a failure to supply nutritive material proper to the needs of the body. Too rapid growth is capable of causing anæmia, the frame seeming to outgrow the quantity of blood manufactured, just as it is apt to become too large for the functional capacity of certain organs. In addition, we must recognize the fact that in some individuals a condition of anæmia seems to be a constitutional characteristic, and to be not incompatible with a fair degree of health. Malaria, as a cause of anæmia, seems to act with even greater intensity in children than is the case in adults, while the anæmia of acute rheumatism at times reaches an extreme grade. Further than in these respects the secondary anæmia of childhood differs in no way etiologically from that in adult life.

Symptoms.—The general appearance of a child with simple anæmia is too well known to require description. The white skin, pallid mucous membranes, waxy appearance of the nails, and blueness of the white of the eye are seen in children as plainly as in adults, if not more so. The subjective symptoms of anæmia do not attain much prominence in childhood, as not only is the child less well able to express its sensations than is the adult, but also because it simply ceases to play around or to exert itself when it feels the subjective sensations produced by anæmia, instead of being compelled, as is the adult, to struggle against discomfort in the endeavor to continue the duties of life.

One of the most frequent symptoms observed in children is the tendency to syncopal attacks. These may occur apparently causelessly, or may be readily induced by violent emotion, slight pain, or confinement in a poorly ventilated apartment. Shortness of breath upon exertion is also frequently present, although in children too young to feel the stimulus of competition this may be shown merely by an indisposition to exertion. Rarely, except in cases of extreme degree, is any œdema discoverable.

The hæmic murmur at the apex or base does not seem to be produced in children so readily as is the case with adults.

The examination of the blood shows a reduction in the red blood-corpuscles, with a corresponding diminution of hæmoglobin; that is to say, the *valeur globulaire* does not differ from the normal. In extreme cases poikilocytosis may be observed. A relative increase of white blood-cells as compared to the red may be present, owing to the reduction in number of the latter.

Diagnosis.—There is, as a rule, no difficulty in determining the existence of simple anæmia, but the diagnosis cannot be considered as complete until the cause of the poverty of the blood has been detected. The question of the causative factor in simple anæmia of the young requires not only a careful examination of the child itself, but a minute scrutiny of all of the hygienic surroundings.

The differential diagnosis between simple, secondary anæmia and that of chlorosis and of pernicious anæmia is readily made by an examination of the blood. In simple, secondary anæmia blood-corpuscles and hæmoglobin are reduced together, and to an almost equal extent, whereas in chlorosis the hæmoglobin reduction far exceeds that of the corpuscles, and in progressive pernicious anæmia the corpuscular poverty exceeds that of hæmoglobin. From splenic anæmia the diagnosis must be made by the detection of a cause other than the enlarged spleen.

Prognosis.—This depends entirely upon the cause. The anæmia itself rarely reaches a degree sufficient to cause anxiety.

Treatment.—While removal of the cause, when possible, is the prime object of treatment, we may frequently combine our symptomatic treatment of the anæmia with the hygienic and medicinal treatment of the previous affection. Good, nourishing food in quantity and quality to suit the age of the patient and the condition of the digestive organs, abundance of fresh air, and an amount of exercise adapted to the primary disease and to the strength of the patient are all-important aids in treatment.

For the purpose of increasing the richness of the blood in corpuscles and coloring matter we have two drugs upon which reliance can be placed, iron and arsenic. In employing iron it is important to remember its marked tendency to interfere with digestion, and in cases dependent upon gastro-intestinal disturbances we can frequently increase the lacking blood-elements more rapidly by first correcting the digestive troubles, when, indeed, the iron may not be

required at all. The best forms for its administration to children are the syrup of the iodide of iron, reduced iron, or one of the vegetable salts of iron. The dose of whatever preparation may be selected should be carefully regulated to the age of the patient, and the drug should be discontinued or its amount lessened when it produces constipation or when the stools are distinctly darkened. In this form of anæmia it is unwise to give more iron than can be absorbed and utilized, whereas in chlorosis even the iron that is voided with the feces seems to have been of some utility.

Arsenic is of great value as a restorer of the red corpuscles, probably by its action upon the blood-making organs. It is pre-eminently useful in the anæmia of chronic malarial poisoning, and is of marked value in the later treatment of mucous disease with anæmia. It is often well to combine iron and arsenic, as they seem to virtually assist each other in many cases; some such form as the following may be employed:

R. Liquor. potassii arsenitis ℥j.
Syrup. ferri iodidi ℥ix.—M.

Sig. Ten drops thrice daily.

THE PRIMARY ANÆMIAS.

CHLOROSIS.

WHILE essentially a disease of youth as opposed to childhood and infancy, this disease is occasionally met with before the former period of life is reached. It is therefore proper that it should find a place in a work upon pediatrics.

Etiology.—While much has been written upon the essential cause of this condition, it cannot as yet be said that the etiology is by any means definitely settled. The theories regarding it are too numerous to be even enumerated. The most satisfactory explanation is that the excessive destruction or imperfect formation of hæmoglobin is due to either the defective absorption and assimilation of iron from the intestinal tract or to the absorption from the bowel of poisonous principles with hæmolytic properties. The view advocated by Virchow that it is caused by congenital hypoplasia of the vascular system, and the view that it depends upon developmental imperfection of the genital apparatus, cannot be considered as tenable considering the rapid and complete cure following the employment of proper hygienic and medicinal treatment.

Age is an etiological factor of great importance, most of the cases occurring between the thirteenth and twentieth years of life. Instances have been observed, however, in individuals even below the former age.

Sex has a strong determining influence, the vast majority of cases occurring in females, and but light grades of the affection being seen in boys. Heredity cannot be said to have any but a predisposing influence, and even that is doubtful, although Trousseau and others claim that the disease is very frequent in tuberculous families.

Habits of life play an important part in its production, the overworked with but little opportunity for the enjoyment of fresh air, exercise, and mental relaxation being those most frequently affected. Depressing emotions, sexual abuse, and fright seem to act as causes, either directly or remotely. The menstrual disturbance so frequently seen in connection with this particular alteration in the composition of the blood must be looked upon as a result rather than as a cause.

Symptoms.—The complaint that induces a patient with chlorosis to seek

medical advice is variable. Sometimes it is the shortness of breath upon exertion, at times the interruption of the menstrual periods, and at times the cephalalgia. The usual history given is that the patient has suffered from vertical headache for a variable time, with shortness of breath upon exertion, palpitation, marked lassitude, and frequent fainting-spells. The date of appearance of the several subjective sensations is as variable as is their relative intensity. The symptoms above enumerated are those most constantly present. Constipation is usually marked, and a desire for unnatural articles of diet is at times a prominent feature. Gastralgic attacks are frequently present.

The appearance of the patient is extremely characteristic. The skin has a peculiar olive tint, which, taken in connection with the pale lips, is imitated by no racial peculiarities of coloring. There is apt to be a certain ashy appearance about the angles of the mouth. The expression is usually languid with an appearance of sadness, while the features frequently show some heaviness of outline. There is a variety of chlorosis, first described by Wendt, wherein the cheeks retain an abnormally red color—*chlorosis florida seu rubra*. Occasionally a deposit of pigment in the neighborhood of joints is observed. The mucous membranes are pallid to a varying degree according to the extent of the anæmia. There may be slight puffiness beneath the eyes, and the feet or ankles may show slight œdema with but little pitting upon pressure. Marked œdema is, however, rare. There may be visible pulsation of the vessels of the neck. The subcutaneous fat is seldom decreased; in fact, the condition of *embonpoint* is that most frequently seen. The pulse is usually rapid and compressible. The apex-beat of the heart is usually plainly visible, and more diffuse than in health. Auscultation reveals, in all marked cases, a soft blowing murmur at either the apex or base, or both, with sharply-defined and somewhat valvular first sound. Over the veins of the neck there is almost always to be heard a loud venous hum. Thrombosis is apparently rather favored by the condition of the blood.

The examination of the blood is of itself sufficient for a diagnosis. The characteristic change is a marked decrease of the percentage of hæmoglobin. With a corpuscle count of 4,500,000, or even over 5,000,000, per cubic millimetre the hæmoglobin may be decreased to 50 or 40 per cent. of the normal. Less characteristic appearances are the pallor of the drop as it flows from the finger and the variety in the size and shape of the red blood-cells when seen through the microscope.

The genital apparatus is usually said to be undeveloped. I have, however, seen within the past year a chlorotic, aged fifteen years, with mammæ, areolæ, and nipples of the size and appearance of those seen in adult life. The urine presents no changes of note save in that it is of low specific gravity and pale in color, contrasting strongly with the low specific gravity and dark color of the urine in cases of pernicious anæmia. Albumin in small quantities is occasionally found.

Morbid Anatomy.—There have been no distinctive lesions found in the few fatal cases that have come to autopsy. The narrowness of the arteries with the small size of the heart noted by Virchow, and the presence in some cases of a poorly-developed uterus and its appendages, are all that have been noted aside from the apparent bloodlessness of the organs and the retention of a fair amount of adipose tissue. In some cases the left ventricle has been dilated. No alterations in the blood-forming organs have been reported.

Diagnosis.—As has been said, the appearance is characteristic. The tint of the skin is quite different from the yellowish-brown stain of jaundice and from the lemon-yellow tint of pernicious anæmia and the cachexiæ. The

sclerotics are of a clear blue color, in contradistinction to the yellowish coloration of icterus.

From these as well as other diseases the blood-examination will separate this affection at once. From pernicious anæmia and Bright's disease the absence of retinal disturbances would readily distinguish it; while in the former the examination of the blood is as characteristic as it is in chlorosis, and in the latter the presence of tube-casts and absence of oligochromæmia are points of plain significance.

Prognosis.—The outlook is extremely favorable, providing only that patients can be persuaded to continue treatment until absolute cure is established. The tendency to relapse is very marked, and patients frequently cease their visits when their most marked symptoms have been relieved, only to return in their former condition after the lapse of a few weeks. The disease is but very rarely fatal, and the unfavorable result is due to the onset of some incidental affection. The only complication of note is gastric ulcer, and this is seen but rarely. Permanent disease of the heart may result in protracted cases.

Treatment.—This is most satisfactory if the patient persist in treatment until cure is complete.

Hygiene plays an extremely important part. Plenty of fresh air, with moderate exercise and a plain but nourishing diet, will do much to hasten the cure. In some cases absolute rest in bed with milk diet seems to act well, particularly in the more severe and obstinate cases.

The daily use of the flesh-brush upon rising in the morning is of value, not only in relieving the coldness of the extremities that is often present, but in improving the general nutrition. In vigorous subjects cold sponging before breakfast will help to increase the general tone of the system. The bowels must receive careful attention. Daily evacuations should be procured by regulation of the diet, the use of "cannon-ball" massage to the abdomen, and, if necessary, by the use of tonic laxatives. Of the latter, the best by far is aloës or aloin. The latter may be made up into a pill with extract of *nux vomica* and extract of belladonna, and should be taken at bed-time. The pill of aloës and myrrh of the United States Pharmacopœia is an excellent combination for older subjects.

The specific remedy for the disease is iron. The simpler the form in which it is given, the better. The most satisfactory is in the combination known as Bland's pill (*R. Ferri sulphat. exsicc., Potas. carb. (pur.), āā gr. iij.*). This may be given after meals, increasing from one to three times a day, to two pills three times daily in the first ten days, and maintaining or even increasing this number until the hæmoglobin has reached the normal amount. Where objection is made to taking pills, as is frequently the case among the class in which this disease is most prevalent, powdered iron may be readily given. The great point is to give the drug steadily and unremittingly until the oligochromæmia has been absent for one or two weeks or even longer.

PROGRESSIVE PERNICIOUS ANÆMIA.

THIS is an intense, generally progressive, alteration of the blood arising spontaneously, characterized clinically by the symptoms and signs of marked anæmia, by diminution of the number of the red blood-corpuscles without corresponding decrease in the amount of hæmoglobin, and by an almost invariably fatal result.

The name of this condition must be looked upon as being provisional. It

is probable that in the future some more definite knowledge may be obtained that will enable us to separate the cases now grouped together under the above title into separate classes depending upon etiological factors that are at present unknown. Formerly cases were grouped under this title that are now known to be separate pathological processes, of which the anæmia was merely a symptom, notably those of atrophy of the gastric mucosa and those due to intestinal parasites. At present, however, we must include under one name a class of cases that have no apparent causation in organs other than those immediately concerned in blood-formation, and which still present a uniform grouping of symptoms.

Etiology.—The actual cause of this disease is as yet unknown. The researches of Quincke and Peters upon the excess of iron found in the liver of patients dying of it, and the observations of Hunter upon the dark color of the urine from the presence of pathological urobilin, would point to the existence of some cause for an increase of hæmolytic. Whether this be a poison created within the body has not as yet been proven, but from the remarkable resemblance between this and the anæmia from atrophy of the stomach it is at least possible to suppose that the hæmolytic may be produced by the absorption of some toxic principle from some portion of the alimentary tract.

Age is a marked etiological factor, inasmuch as the large majority of cases occur during middle life. That it does occur in young persons with moderate frequency is shown by the fact that cases have been collected by Griffith,¹ wherein the disease has occurred at the ages of sixteen months, three, five, seven, eight, ten (2 cases), eleven (2 cases), twelve, fifteen, and eighteen years, and in one other boy in which the age was not given; while I have found additional cases reported as pernicious anæmia, without an exhaustive search of the literature, at ages of eleven months,² one year and four months,³ two,⁴ four,⁵ eleven (2 cases),⁶ thirteen,⁷ fifteen,⁸ sixteen,⁹ seventeen,¹⁰ and twenty¹¹ years.

The female is rather more prone to the disease than is the male sex.

In one of Escherich's cases the appearance of the disease followed close upon vaccination with animal lymph, but whether there was any relation between the two events it is impossible to say.

Symptoms.—The most striking subjective symptom is extreme and progressive weakness. Shortness of breath and vertigo soon become prominent symptoms. While feeling extremely ill, the patient retains a fair amount of fat, and save for extreme pallor has the appearance of a well-nourished individual.

The weakness and pallor increase gradually with, at times, temporary short intervals of apparent improvement. Dyspnœa increases, the extremities become œdematous, and the patient is at length compelled to remain in bed,

¹ Keating's *Cyclopædia of Diseases of Children*, 1890, vol. iii. p. 809.

² D'Espine and Picot (*Revue de Méd.*, 1890, p. 859): blood-count not given, doubtful.

³ *Ibid.*: blood-count not given, probably a true case.

⁴ Escherich (*Wiener klin. Wochenschr.*, 1892, No. 13, p. 193).

⁵ Mott, *Practitioner*, Aug., 1890.

⁶ Ashby and Wright (*Diseases of Children*, 1892, p. 337): no blood-count, urine of low spec. grav., and pale, therefore doubtful.

⁷ D'Espine and Picot (*loc. cit.*): no blood-count given.

⁸ Taylor (*Guy's Hosp. Rep.*, 1878): doubtful, no blood-count.

⁹ Wilks (*Guy's Hosp. Rep.*, 1857, p. 203): probably a case of pernicious anæmia, though described as a case of "idiopathic fatty degeneration."

¹⁰ Handford (*Br. Med. Jour.*, 1891, p. 445).

¹¹ Roosevelt (*N. Y. Med. Record*, 1888, p. 407).

the whole body being sometimes water-logged with anasarca. Occasionally irregular elevations of temperature arise without apparent cause. Gastro-intestinal disturbance may be present, but in cases unassociated with gastric and intestinal atrophy they sink into insignificance in comparison with the intense languor and shortness of breath. Hæmorrhages from the mucous membranes and beneath the skin are sometimes present and may be profuse.

As a result of treatment or without apparent cause, the condition may for a time improve, but the course is usually progressively downward until death occurs from simple asthenia, possibly hastened by an attack of intestinal disturbance or by the onset of some acute inflammatory trouble.

The appearance of the patient is almost pathognomonic. The skin is of a peculiar pale-lemon tint, the lips almost white, the conjunctivæ of a pearly whiteness. Areas of pigmentation may be present on various parts of the body. The retention of a fair degree of *embonpoint* with the extreme pallor at once suggests this disease to one who has seen a case thereof.

Upon physical examination nothing abnormal may be found save soft hæmic murmurs at the apex or pulmonary cartilage and venous murmurs in the neck. The pulse is soft, readily compressible, and gives an impression to the finger similar to that of aortic regurgitation, which disease this also somewhat resembles in the occasional presence of a capillary pulse. The urine is peculiar in that with low specific gravity the color is quite decided—due, according to Hunter, to the presence of pathological urobilin. Upon ophthalmoscopic examination streaks of hæmorrhagic extravasation are frequently to be seen.

The examination of the blood is of itself sufficient to determine the diagnosis. The blood as it exudes from the finger is usually of a paler color than normal, and may be obtained only with great difficulty. Upon examining a fresh specimen there is found to be extreme irregularity in the size and form of the red cells. There are seen in the same field numerous red cells smaller than the normal, side by side with others of double the size of the latter. Nucleated red cells of large size are also seen. There is little tendency to the formation of rouleaux. The red blood-cells are far below the normal average per cubic millimetre. Their number varies much with the duration and severity of the individual case: it may sink to below 500,000 per cubic millimetre. The estimation of hæmoglobin shows that this is in excess of the amount corresponding to the cellular reduction. This disproportion of the number of red cells and the amount of hæmoglobin is characteristic of the disease—the *valeur globulaire* is exceedingly high.

Morbid Anatomy.—The skin is generally of a markedly yellowish-white color. The subcutaneous fat is usually remarkably well preserved and is of a light-yellow color. The muscles are peculiarly red, in marked contrast with the pallor of other tissues and of the muscular tissue in other forms of anæmia. All of the internal organs look blanched, but upon the various serous membranes ecchymotic areas are frequently seen. Punctiform hæmorrhages may also be present in the skin, mucous membranes, connective tissue, muscles, heart-wall, bone-marrow, lymph-glands, spleen, liver, pancreas, lungs, and dura mater. They are due, according to Bermer, to fatty degeneration of the capillaries, although other observers have failed to find the change described. In the serous cavities a varying amount of clear serum is present. The heart is usually large and soft, its walls flabby, its chambers almost empty of blood. "Tabby-cat mottling" of fatty degeneration is frequently present, or the whole tissue may be pale and fatty-degenerated. The spleen shows no constant changes. The gastric mucosa may be found atrophied in some cases of appa-

rently true idiopathic pernicious anæmia; but these cases should not be classed under the name of the disease under consideration unless the view that atrophy of the gastric and intestinal glands is one of the results thereof. The liver is fatty, and shows the only really characteristic change of any of the organs. Upon microscopic examination there is found an excess of free iron in the cells of the outer and middle zones when the sections are treated with proper reagents. The kidneys may be the seat of marked fatty degeneration, and iron has been occasionally detected in the renal cells. The marrow of the shaft of the long bones is of a deep brick-red color, resembling the foetal condition, but the appearance is not characteristic, as it has also been found in other forms of anæmia. In the posterior columns of the spinal cord there has been found a process resembling in every respect that seen in locomotor ataxia.

Diagnosis.—The chief difficulty in diagnosis lies in the exclusion of a primary cause for the anæmia. The appearance of the patient, the subjective symptoms, and the progressive course will usually lead to a correct diagnosis. An examination of the blood definitely decides the question. The diseases which most resemble pernicious anæmia are atrophy of the gastric tubules and malignant disease of the internal organs, particularly those of the digestive tract. Careful examination will usually exclude the latter even without an examination of the blood. Certain cases of atrophy of the gastric tubules have so resembled pernicious anæmia as to render a distinction between them an impossibility. Unfortunately, in these cases the chemical examination of the gastric contents is of but little aid, as Ewald has found that hydrochloric acid is absent from the gastric juice in pernicious anæmia as well as in gastric atrophy, and the peptonizing power is diminished in both conditions.

Prognosis.—The outlook is extremely grave. As a rule, death comes in spite of all our efforts. A fatal result occurred one month after the first appearance of pallor in the two-year-old child reported by D'Espine and Picot, and in Kjellberg's case of a boy aged five years death occurred six weeks after development of symptoms. Recovery may be considered impossible if the red cells number 500,000 per cubic millimetre or less. Since the discovery of the value of arsenic in this disease the prognosis is somewhat less hopeless than formerly. By its use apparently hopeless cases may be at least temporarily relieved. Too often, however, the improvement is but temporary, and relapse soon takes place. Death comes from exhaustion or from the onset of some intercurrent disease. A sharp attack of diarrhoea or an inflammatory disease of the respiratory tract is frequently the immediate cause of death. Hæmorrhage is rarely of sufficient amount to cause death. Litten reports a case that apparently passed into leukæmia.

Treatment.—Absolute rest with freedom from worry and excitement is of prime importance. A diet selected with care and adapted to the needs and capacity of the individual is to be directed.

Among drugs none can equal arsenic in value. By its means the number of red blood-cells may be increased to within a fair degree of normal, and with corresponding amelioration of symptoms. It should be given freely up to the point of tolerance. It is better to begin with small doses well diluted, and to increase as rapidly as is consistent with the avoidance of toxic symptoms: upon the appearance of gastro-intestinal disturbance or of œdema either the use of the drug should be entirely discontinued for a time or the dose should be much reduced. The pigmentation occasionally seen in the course of the disease should not cause needless fear of arsenical pigmentation. Iron is but seldom of value. It may, however, be used in cases showing an intolerance to arsenic. Rectal

injections of blood prepared in various ways are no longer considered worthy of the hope that was at one time placed in them.

The inhalation of oxygen may relieve the dyspnoea that is at times severe, but nothing more than palliation can be expected to result from its use.

If the theory of intestinal absorption of ptomaines in the causation of this disease be correct—and there seem many reasons for believing it to be so—rendering aseptic the intestinal canal would be a rational means of cure. It is well, therefore, to keep the bowels opened regularly, and to administer in appropriate quantities salol, thymol, or β -naphthol in order to accomplish what we can in this direction.

SPLENIC ANÆMIA.

IN a considerable number of children there is found a marked degree of anæmia associated with no appreciable lesion save enlargement of the spleen. Rendu has reported a case wherein, after the lapse of two years, an increase in the number of white blood-corpuscles occurred, and Gilbert saw a case that later was transformed into lymphatic leukæmia.

Etiology.—Much discussion has been indulged in as to the cause of this form of anæmia in childhood, and even now it cannot be said that any uniformity of opinion has been obtained. Malaria is certainly capable, when long operative, of producing both anæmia and chronic splenic enlargement in children, just as in the case of adults. The cases presenting a malarial history comprise, however, but a very small minority of the cases in which this affection has been observed.

The two diseases that appear to have most claims as etiological factors are rickets and inherited syphilis. Out of 30 cases, Carr found 27 with other distinctly rachitic lesions; in 14 cases syphilis played at least a prominent part. In 60 rachitic children Kuttner found a palpable spleen in 44, in 33 of which the organ was markedly enlarged. In only 2 of the 60 cases was there a clear history of syphilis, but in 13 there was a history that the mother had had miscarriages or stillbirths. In 63 cases examined by them, Fox and Ball found that rachitic symptoms were present in almost all; and in one series of 105 consecutive cases of rickets the spleen was enlarged in 14 per cent.; in another series of 84 cases of very marked rickets, enlargement was present in 40 per cent. That inherited syphilis may be more than a predisposing factor is rendered highly probable from further statistics furnished by the last-named authors. In 63 cases of enlargement of the spleen with anæmia they found inherited syphilis in 41 per cent.; while in 155 cases of inherited syphilis the spleen was enlarged in 48.4 per cent. The influence of hereditary syphilis in causing rickets should not be overlooked, and it seems more than likely that the most potent factor is rickets. It is interesting in this connection to learn that Sutton (according to Fox and Ball) has found both liver and spleen constantly enlarged in monkeys, where rickets is produced by causes other than syphilitic taint.

The disease would appear to be frequently found in members of the same family, partly due, no doubt, to the fact that the individuals were all subject to the same conditions of life.

Boys are more often affected than girls, Kuttner having found it in 37 boys out of 60 cases. The disease has been seen at the age of two months (Carr) and in adult life, so that no definite statement can be made as to age as a predisposing factor.

Pathological Anatomy.—The only characteristic lesions found relate to the spleen. The organ is enlarged, the capsule thickened and adherent, the

parenchyma firm, with marked increase of fibrous tissue. The microscopic examination shows increase of fibrous tissue, with atrophy of Malpighian bodies and disappearance of adenoid tissue (Peter). The marrow of the long bones may have become lymphoid in character. In the other organs various changes are to be found as coincidental affections. These are practically the lesions discovered after death in children with rickets or inherited syphilis. The most frequent abnormal conditions found relate to the respiratory organs. There may be bronchitis, atelectasis, pneumonic consolidation, or the deposition of tubercles. The gastro-intestinal tract may show the lesions of a chronic catarrhal inflammation.

Symptoms.—Lassitude and general weakness on the part of the child may be the causes of medical treatment being sought. In other cases the peculiar pallor may have called the attention of the parents to the child's condition. The enlarged spleen may have caused anxiety, or the child may have been brought for treatment on account of the catarrh of the respiratory or digestive tract that is a frequent accompaniment of the condition. The existence of the disease may be discovered accidentally in examining a child presenting other manifestations of rickets. The complexion is of a peculiar waxy, pallid hue, with rather a muddy tint. The mucous membranes are blanched, the tongue pale and flabby.

Upon examination of the trunk there are found in rachitic children not only the prominent abdomen that is usually seen in children of this class, but there may be visible tumor in the hypochondriac and lumbar regions of the left side. Frequently the enlargement of the spleen may not be discovered until palpation reveals a resisting mass. In marked cases the spleen can be readily felt as a sharply-defined solid tumor, with its anterior edge notched in one or two places. The organ can be made more prominent by pressure with the free hand upon the left hypochondriac and lumbar regions. In less well-marked cases careful palpation, with firm pressure upon the left flank, may be required in order to bring the anterior edge forward sufficiently to be felt through the abdominal wall. Testi heard a vascular murmur over the enlarged spleen.

Examination of the blood reveals a reduction in the number of red corpuscles. Kuttner found the number in 10 cases to vary from 1,020,000 to 4,080,000, with a hæmoglobin value of 35 per cent. in the former instance and 73 per cent. in the latter. There is no absolute increase in the number of white blood-corpuscles, although in fatal cases there may be at times an increase in these elements toward the close of life.

Irregular fever is frequently present, possibly owing to the frequent catarrhal complications. In some cases epistaxis may be present, in some subcutaneous hæmorrhages. Albuminuria seems to be rare, although Carr found it present in two of his cases. The liver is frequently enlarged, and there may be some enlargement of the deeper sets of lymphatic glands. Catarrhal inflammation of the bronchial mucous membrane and in the gastro-intestinal tract is frequent, but it is impossible to attribute it to the condition of splenic anæmia, owing to the frequent coexistence of the rachitic condition.

Diagnosis.—When the spleen is much enlarged the history of the case and the examination of the blood render the diagnosis a matter of ease. The absence of increase of white blood-cells would differentiate the disease from splenic leukæmia, and an examination of the blood for the plasmodium malarie would cast out malarial enlargement. From enlargement of the spleen from amyloid infiltration the absence of a history of the influences causative of that affection, and the failure of evidence of a similar infiltration of the liver and

kidneys, would differentiate this disease. From an enlarged left kidney the diagnosis is to be made by the presence of notches in the anterior border, by the direction of enlargement, by the greater motility of the tumor upon bimanual examination, and by the absence of urinary changes. The acute enlargements from typhoid fever, embolic abscess, and acute malarial poisoning are readily excluded by the history of the case. Enlargement from cirrhosis of the liver would be but little apt to cause embarrassment in arriving at a diagnosis.

Prognosis.—While fatal cases are not rare, the prognosis is not, as a rule, bad if proper hygienic conditions can be enforced. Of Carr's 30 cases, 10 died, 6 disappeared from sight, 13 recovered, and 1 remained stationary. The chief cause of death is the occurrence of acute respiratory or digestive inflammatory complications.

Treatment.—Of prime importance is the securing of proper hygienic surroundings. Plenty of fresh air, well-ventilated sleeping apartments, and a proper amount of outdoor exercise are essential. The diet must receive careful attention. The food should be plain and nourishing, with absence of excess of farinaceous articles. The clothing also should be regulated.

Of drugs, cod-liver oil, arsenic, and iron are the most useful. Phosphorus may be used in those markedly rachitic. In cases that have a distinct history of inherited syphilis mercury may be given, but even in the manifestly syphilitic the splenic enlargement is apt to undergo no diminution from its use. The judicious administration of cod-liver oil by either internal means or by inunction, or by both methods combined, with the use of a combination of iron and arsenic, such as was mentioned in the section upon Secondary Anæmia, will be found to be the best line of treatment in connection with careful correction of insanitary conditions.

The application of electricity over the spleen may produce lessening in the size of the organ.

LYMPHATIC ANÆMIA.

THIS affection is a more or less generalized condition of the lymphoid tissue of the body, characterized by enlargement of groups of glands or increase in the normal lymphoid structures of a part, accompanied by oligocythæmia and a varying amount of enlargement of the spleen.

The disease bears in many respects a close resemblance to the lymphatic form of leukæmia, and, in fact, the leucocytosis that frequently is present to a marked extent has been seen to pass into a condition of true leukæmia. The whole subject of the relation between these two diseases of the lymphoid tissues of the body, and also between them and diffuse sarcomatous disease of the lymphatic glands, still needs further study, in spite of the work that has already been done in attempting to assign them to their proper position.

Etiology.—This is still far from decided. Inherited syphilis has been supposed to play a certain rôle, but it is doubtful whether the association has been more than a coincidence. Age certainly exerts some influence, as the disease is very common in the young. Males are more frequently attacked than females. Heredity has not been shown to exert any influence. The action of continued local irritation or inflammation would seem to be a strong etiological factor, and it may be owing to the frequency of long-standing lesions of the skin, of the face and head, of the jaws and ears, that the cervical chains so frequently are the earliest and most markedly involved groups.

Symptoms.—The disease begins insidiously with enlargement of some group of lymphatic glands, with increasing anæmia with its accompanying

subjective symptoms, and with progressive weakness. The glands most frequently attacked are those in the posterior cervical triangle, but the axillary or inguinal glands may be first involved. Deeper sets of glands, as those in the thoracic or abdominal cavities, may be involved before the external tumors appear, or even without involvement of the superficial groups. The external glands may form large masses, producing much disfigurement. The cervical glands may obliterate the outlines of the neck or may encircle the front portion of the neck like a collar, and produce marked dyspnœa. The axillary group may be enlarged sufficiently to prevent the apposition of the arm to the side, while the inguinal glands may enlarge sufficiently to embarrass locomotion. Pressure of these masses may produce various secondary results, such as pain radiating down the trunks of the nerves running near to the tumors, and œdema from pressure upon the venous trunks. When the visceral sets of glands are involved, there may be no outward signs of their presence, although the retroperitoneal and mesenteric groups may be enlarged so much as to be both seen and felt. By pressure upon various organs, blood-vessels, or ducts they may produce effects varying with the part involved. Dyspnœa may be produced from pressure upon the bronchi; cyanosis or œdema of the face from pressure upon the superior vena cava. Dyspeptic symptoms, constipation, anuria, ascites, and œdema of the lower extremities may be caused by enlargement of the groups within the abdominal cavity. Secondary involvement of the spinal cord may produce paraplegia from pressure.

The lymphoid tissue in the tonsils, tongue, pharynx, skin, and intestinal wall is occasionally the seat of the same outgrowth, producing symptoms varying with the situation involved.

Either continued mild pyrexia, alternating periods of pyrexia and apyrexia, or distinctly intermittent fever is usually present during some period of the course.

The general symptoms are those due to the anæmia. Vertigo, headache, lassitude, and dyspnœa may be obtrusive symptoms. The patient is usually very pale, and the white skin with thickened neck forms a picture that could with difficulty fail to suggest the presence of this disease.

The examination of the blood shows a decrease of the number of red blood-cells to a varying degree. Poikilocytes are common, and nucleated red blood-corpuscles are occasionally seen. There is leucocytosis, which in some cases attains to such a degree that the case must be classed as a lymphatic leukæmia.

The patient usually succumbs after a period varying from less than a year to five years (Gowers) from asthenia. Obstinate diarrhœa may occur at any time, even without involvement of the intestinal canal. Death may occur from pressure upon the air-passages before the general condition of the patient would excite alarm.

Morbid Anatomy.—The skin is pale, the subcutaneous layer of adipose tissue more or less decreased. The post-mortem findings vary much in different cases in accordance with the glands involved. Usually there are masses of enlarged superficial glands in the neck, axillæ, or groins. These are found to be composed either of isolated, enlarged nodules varying from the size of a pigeon's egg to that of a hen's egg, or of masses of lymphatic glands welded together or even infiltrating neighboring structures, from which they may be separated either with difficulty or not at all. Upon section the individual glands present various appearances even in the same case. They may be soft and of a color not differing much from the normal, and may yield an abundant milky juice, or they may be hard and firm, showing a clear white color of the cut surface without any juice.

Any of the lymphatic glands in various parts of the body may be involved in the same way. The groups of glands in the mediastina, the bronchial glands, the retroperitoneal, or the mesenteric, may each or all of them be enlarged and more or less matted together. The thymus gland has been found either uniformly enlarged or the seat of lymphoid tumors.

The spleen is enlarged in the great majority of cases, either from simple hypertrophy or from the presence of tumors of lymphoid tissue. The liver and kidneys may show nodules of lymphoid tissue. The lungs are sometimes affected from encroachment of growths from the bronchial group of glands or by the growth of independent foci of lymphoid tumors. The heart rarely shows similar growths in its substance.

Various secondary morbid changes are produced by the pressure of the masses of glands upon neighboring structures.

The marrow of the long bones may have a puriform appearance or may be of an intense red color.

Histologically, the lymphoid tissue of the enlarged glands and of the isolated tumors is found to be composed of a delicate reticulum enclosing round cells. In some glands there is also an increase of fibrous tissue.

Diagnosis.—In many cases it is impossible to state whether the case in hand should be classed as one of pseudo-leukæmia or as a true lymphatic leukæmia. In the latter disease the spleen more frequently attains a considerable size than in the cases now classified as pseudo-leukæmia. As this disease may pass into a true leukæmia, in so far as the blood-estimation forms a criterion, and as the treatment is practically the same for the two affections, the differential diagnosis makes but little practical difference. The name “pseudo-leukæmia” should, however, be applied only to those cases wherein the proportion of white to red cells does not exceed one to thirty.

From tubercular adenitis, the so-called scrofulous enlargement of the glands, the differential diagnosis must be based partly upon the family and past personal history, partly from the appearance of the patient, but chiefly from the more localized character of the glandular swelling and the tendency to caseation and suppuration in the tubercular disease.

Secondary involvement of the lymphatic glands by cancer will not enter into consideration in those below adult life.

Prognosis.—The outlook is extremely unfavorable. The progressive tendency of the disease may sometimes be combated by treatment, but cure can be expected but rarely. In the early stages, where the involved glands are accessible to the surgeon, the disease may be cured by operative treatment. The degree of asthenia and the extent of the anæmia offer some means of forming a prognosis as to duration.

Treatment.—In early cases, where superficial glands are alone attacked, the chance of cure by surgical means should not be neglected. In cases of doubtful nature, where the diagnosis between this affection and an essentially local disease of the affected glands is difficult, the safest course is to avail ourselves of surgical means of cure. Of drugs, arsenic is the only one upon which dependence can be placed. It should be administered in ascending doses until the point of tolerance is reached. Iron is of secondary value as a hæmatonic, but may be combined with arsenic, preferably in the form of the officinal syrup of the iodide of iron. External applications to the affected glands can only be of value where the integrity of the skin is in danger.

Tracheotomy may be necessitated by pressure upon the trachea or if the enlarged glands interfere with the nerve-supply of the vocal cords.

LEUKÆMIA.

LEUKÆMIA is a disease of the blood-making organs, characterized, clinically, by the symptoms of anæmia, excessive increase in the number of white blood-cells, and a tendency to hæmorrhagic extravasation; pathologically, by enlargement of the spleen and lymphatic glands and by changes in the bone-marrow, either separately or in combination.

The condition of the blood in this disease is mimicked in health after eating (physiological leucocytosis) and in various organic diseases wherein there is an intense local lesion (pathological leucocytosis), as in pneumonia, empyema, etc. The term "leukæmia," however, must be limited to cases wherein leucocytosis is more or less constant, is of marked degree, and is associated with the characteristic lesions of spleen, lymph-glands, or bone-marrow.

As to the nature of the disease there is much diversity of opinion. The term "leukæmia" is at present the most applicable, because non-committal, name that we can apply to it.

Various divisions have been made in respect to the part chiefly or solely involved in the disease—splenic, lymphatic, or medullary (myelogenous). Rarely is any one form present alone, but the cases usually fall into the classes lieno-medullary or lieno-lymphatic. Cutaneous, intestinal, and tonsillar forms are curiosities.

The disease bears, in many respects, a close resemblance to sarcomatosis.

Etiology.—The precise etiology of the disease has not yet been decided. It is preceded by malaria and syphilis in a number of cases sufficient to render it possible that these diseases have at least a predisposing influence. Trauma in the splenic region has been followed by its appearance. Some of the more acute cases pursue a course that is strongly suggestive of an infectious origin. Fermi, Powlowski, Bonardi, Kelsch and Vaillard, Klebs, Roux, and others have reported the finding of various micro-organisms in the blood or tissues of cases of the disease. Negative results were reached in Westphal's case in an attempt to obtain cultures from the spleen during life and from the blood and bone-marrow after death. Gilbert unsuccessfully attempted to inoculate healthy dogs with lymphatic glands from a dog affected with the disease. Mosler failed to produce the disease by the injection of leukæmic blood into dogs and rabbits. Bollinger met with a similar result in attempting to produce the disease in healthy animals by the injection of blood from leukæmic animals of the same species. Apparent infection occurred in Obrastzow's experience, where an attendant upon a case died after fourteen days' illness with purpura, hæmorrhages, fever, albuminuria, and a proportion, in the blood, of one white to nine red blood-cells.

The disease is seen at all ages from birth up to the seventy-fifth year. It is most frequent between the ages of thirty and fifty years. It is not rare in childhood, many cases having been reported in infants less than two years of age, while Sängner has reported its existence in a stillborn child. It is more common in males than in females. Heredity has not been proven to be an etiological factor. Horses, oxen, dogs, pigs, cats, and mice suffer from a similar affection.

Symptoms.—The usual symptoms that impel the patient to seek advice are the general weakness, the pallor, the shortness of breath, hæmorrhages from the mucous membranes, the enlargement of the abdomen, or the superficial lymphatic tumors. The disease usually arises gradually, so that, as a rule, marked changes in the organs and blood have occurred before the patient is brought for treatment.

The symptoms produced by the abnormal condition of the blood are similar in the different forms of the disease, but the examination of the patient yields results varying with the type. Breathlessness upon exertion is usually a very marked feature. It may be accompanied by marked vertigo upon change of posture. The bodily strength is impaired to a great degree, but in some cases it is remarkably well preserved in view of the serious changes in the composition of the blood. Hæmorrhages may have occurred from the nose, throat, stomach, or intestines, or there may be hæmorrhagic extravasations beneath the skin. Hæmorrhages in the fundus oculi may produce sufficient interference with vision to attract the attention of the patient. Edes has recorded a case wherein priapism was the first symptom. During the course of the disease occasional rises of temperature may be noted.

Upon examination there is found more or less pallor of skin and mucous membranes. The pulse is soft and compressible, with increased rate. If the anæmia be marked, there may be heard a hæmic murmur over the position of the apex-beat or in the second left intercostal space. The lungs usually present no morbid signs save toward the close of fatal cases, when œdema, congestion, or a fluid accumulation in the pleural cavity may be found. In some cases there is found in the lung what clinically resembles lobar pneumonia, but histologically is found to present features differing from the ordinary form.

Diarrhœa may be persistent, and in some cases a species of dysentery is present. Vomiting is not a frequent symptom. The occasional occurrence of hæmatemesis has been mentioned above.

The urine is usually unaltered save for an increase in the amount of uric acid excreted.

On the part of the nervous system we may have no symptoms. Vertigo and cephalalgia are at times marked. Death may occur from intracranial hæmorrhage. Vision may be much impaired, due to the presence, as revealed by the ophthalmoscope, of retinal hæmorrhages or of leukæmic deposits. Hearing may be impaired. Suchamick has noted a peculiar brownish discoloration of the nasal mucous membrane in one case.

The usual course of the disease is slowly progressive, covering a period of months or years. There have been reported some cases running an extremely rapid course, as in that of Guttman, where a fatal termination occurred after an illness of four and a half days.

The examination of the blood is all-important in determining the nature of the disease. The constant feature is an increase, both relative and absolute, of the white corpuscles. This may attain to an extreme degree, the relative number of white to red cells having even been as two to one in a case reported by Robin. The average ratio of white to red cells is as one to fifty or twenty, in cases without great reduction in the latter elements, as opposed to one to 500 or 700, the average ratio of health. The various forms of white blood-cells are present in different proportions in the lieno-medullary and in the lymphatic varieties. In the former the eosinophilous cells of Ehrlich are the predominant form, whereas in the acute lymphatic variety the lymphocytes form the main proportion of the colorless elements. Where the lymphatic, splenic, and medullary varieties exist together in the same patient, the proportion of the forms of leucocytes will produce variations from the two types mentioned. Myelocytes may be present in large numbers. Charcot's crystals are said to form after the blood has remained upon the slide for a short time.

In the splenic form a prominent feature is the gradual enlargement of the spleen. This occurs to a varying degree, the organ in extreme cases even

reaching to or beyond the median line of the abdomen. The splenic enlargement takes place chiefly in a diagonal direction, downward and toward the right. When the hand is placed over the mass, a rub may be felt and tenderness be elicited by pressure. Spontaneous pain or sense of pressure may be an annoying symptom, while the weight of the organ may produce disorder of digestion or marked constipation.

When the marrow of the bones is affected, there may be tenderness over the affected parts, with localized swellings on the shafts of the long bones or the ribs or sternum.

The lymphatic glands are less frequently involved than is the spleen. The superficial glands show enlargement and can be readily felt, or even seen as isolated groups or chains. The deep glands of the abdominal cavity may be affected.

Morbid Anatomy.—The skin is pale, the subcutaneous fat usually much diminished. The blood has a chocolate color, or may even almost resemble sanious pus. When clotted it has a greenish-yellow color. On the serous membranes there may be areas of hæmorrhagic extravasation. In the serous cavities there is usually an excess of fluid.

The heart is frequently found distended with clotted blood. The lungs present no constant changes, although posterior congestion is often seen. Rarely are there any changes in the thymus gland.

The spleen is almost invariably enlarged to a greater or less degree. Adhesion to neighboring organs is common, explaining the sharp attacks of pain sometimes experienced in the left hypochondriac region. The organ is usually symmetrically enlarged, is of increased density, and on section may show either a brownish color throughout the surface, or there may be scattered areas of a white color due to localized infiltration with lymphoid cells, either in the Malpighian follicles or in the pulp. Hæmorrhagic areas may be present. The spleen may enlarge so rapidly as to cause a rupture of its capsule.

The intestines show at times evidences of lymphoid infiltration, either in the glands of Peyer or in other parts, by thickening without ulceration. The tonsils, pharynx, and stomach have been found to show signs of the overgrowth of lymphoid tissue.

Lymphoid tumors have been found in the liver in sufficient number to notably increase the size of the organ, while the kidneys also may present whitish areas of lymphoid infiltration, as in the case reported by Fränkel. The lymphatic glands of the superficial sets or of deeper parts, as near the root of the mesentery, are in some cases much enlarged, although rarely to so great an extent as in pseudo-leukæmia.

The marrow of the bones is affected in a considerable number of cases, chiefly in conjunction with splenic involvement. In these cases it is found to be of a puriform appearance or to be of a dark-red color. Hæmorrhagic areas may be present. The shaft may be found expanded and the wall thinned. Microscopically, the marrow shows large numbers of nucleated red blood-cells, eosinophiles, and myelocytes.

Diagnosis.—The only diseases with which leukæmia is apt to be confounded are pseudo-leukæmia, splenic anæmia, and scrofulosis. From these the diagnosis may readily be made by an examination of the blood. The numerical increase of the white blood-cells is alone sufficient to make the diagnosis, save in cases of non-leukæmic leucocytosis. From this the diagnosis cannot be made with certainty by the hæmocytometer alone, as in leucocytosis the relative increase of white cells may be greater than in some cases of leukæmia. For the differentiation of these two conditions we may employ the

method of differential staining according to Ehrlich's procedure. While some question has been raised as to the value of the eosinophile cells as diagnostic criteria, this objection cannot now be said to be of weight save in the lymphatic variety, where the cells having eosinophile granules are not present in large number.

Prognosis.—The prognosis as to recovery is grave, although cases have been known to recover. The disease is usually fatal within a few years. In some cases of acute lymphatic leukæmia, as in the case reported by Guttman, death may occur within a few weeks or days.

Treatment.—Rest is of prime importance. The dietary should be selected with care, and should be suited to the digestive power of the individual.

Arsenic is almost the only drug that can be said to be of any real value. It should be pushed up to the verge of tolerance, and its use should be persisted in until either it is evident that no result is being obtained or until the patient is, mayhap, relieved of the disease.

Quinine should be tried in cases giving a malarial history, but it will rarely be productive of much benefit.

Injections of arsenic into the spleen are not likely to materially benefit the patient, and are not without risk. Westphal's case died after a puncture of the spleen for diagnostic purposes, the organ being surrounded by a large blood-clot at the autopsy. Splenectomy cannot be considered justifiable, in spite of Franzolini's successful case, in view of the large mortality attending the operation.

HÆMOPHILIA.

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HÆMOPHILIA is a tendency to obstinate bleeding; inherited; often associated with swelling of the joints.

Etiology.—The hæmorrhages may be traumatic or spontaneous in origin. Certain families are known as “bleeders,” the hæmorrhagic diathesis manifesting itself at any time from early infancy to the end of life. Hereditary transmission takes place mostly through the mother and to her male offspring. If a woman descended from bleeders marry a healthy man, the sons will inherit the hæmorrhagic diathesis, the daughters escaping. In the succeeding generations the sons in whom hæmophilia is manifest will not transmit the diathesis, whereas the daughters, who show in themselves no signs of it, will transmit the diathesis again to their sons. The maternal transmission so continues to many generations, the hæmorrhagic condition appearing in the males, the females escaping, but transmitting the diathesis to their sons. Bleeders usually have large families, some of whom may escape the disease. They are to be found in all localities, in all conditions of life; are healthy in appearance, commonly having fine, soft skins. The Hebrew race is said to be particularly liable to it.

The real cause of hæmophilia is unknown. It is believed that the condition has in some individual instances been acquired.

Pathological Anatomy.—The post-mortem findings do not explain the nature of the affection. An unusual thinness of the walls of the vessels has been observed, though the microscope fails to reveal any essential and constant alterations. The tissues are blanched from loss of blood. Petechiæ and bruised patches are frequently observed upon the surface of the body. The swelling of the joints is due to hæmorrhages into the articulations and the surrounding tissues. Occasionally there is evidence of joint inflammation. At present it has not been determined whether the hæmorrhage is due to some fault in the walls of the vessels, or whether there is some peculiarity in the character of the blood on account of which thrombi are not formed.

Symptoms.—At birth there is nothing in the appearance of the child to indicate the peculiarity of his inheritance. He is usually healthy and bright, and may in the first year develop no signs of hæmophilia. The severing of the umbilical cord does not usually give occasion for obstinate bleeding, and not until his growth and strength lead him into accidents, such as bruises, cuts, scratches, and punctures, does the hæmorrhagic tendency become apparent. Epistaxis is the most common experience which calls attention to the diathesis. This may be acute, obstinate, and alarming. Besides, there may be petechiæ, ecchymoses, hæmatomata, interstitial and external bleeding, traumatic or spontaneous.

A common symptom is swelling of the joints closely resembling rheumatism. It is not uncommon to find hæmorrhage of the gums at the eruption of the second crop of teeth. Slight cuts give rise to troublesome hæmorrhage, slight

blows to marked ecchymoses, and a blister may contain blood instead of serum. Prolonged and dangerous bleedings may follow the extraction of a tooth in spite of the application of the strongest styptics.

The bleeding is from the capillaries, most often an oozing, which may continue from hours to weeks. The subjects of hæmophilia are very sensitive to cold, and suffer from joint-pains apart from those dependent upon hæmorrhage. Such patients pass through the exanthemata and other diseases of childhood without special dangers, and have no marked proneness to phthisis. Sloughing and gangrene are not uncommon accidents of this condition.

Prognosis.—From the nature of the disease it must be considered a constant menace to life. However mild the tendency in the infant, the prognosis should be considered very serious. Of 152 cases of hæmophilia traced by Grandidier, more than half died before completing the seventh year, and only 19 attained majority. The exhaustion of repeated hæmorrhages, or, more commonly, the draining away of blood by continued oozing, may destroy life. The most difficult of control and the most frequently fatal are the hæmorrhages following extraction of teeth or from epistaxis.

There are examples of bleeders who have attained a good age and led busy lives. To this class belongs a very busy practitioner of the writer's acquaintance, who is never without fresh petechiæ of the face, and constantly carries a large red handkerchief for accidental epistaxis.

In females the prognosis is good, neither menstruation nor childbearing being complicated by this capricious example of atavism.

Treatment.—Prophylaxis avails somewhat to diminish the accidents of hæmorrhage. The system may be fortified by abundant fresh air and tonics, by judicious exercise and general hygiene. The child should be guarded, so far as possible, from bruises, cuts, and punctures. Vaccination, though not historically accounted a dangerous procedure in bleeders, should be accomplished rather by scarification than by incision. Slight operations should be seriously considered before they are undertaken, and every needed means of hæmostasis should be at hand. The extraction of teeth should be avoided. Nearly every practitioner has had at least one trying experience with obstinate hæmorrhage from such cause in a person not hæmophilic, and can well understand the importance of this advice.

It is well to have the diet properly regulated for hæmophilics, giving vegetables and generally wholesome mixed meals, without excess of meat. The bowels should be regulated so as to correct any tendency to a "full-blooded" condition. Where premonitory symptoms indicate an impending hæmorrhage, it is well to relieve the bowels by a mercurial purge, followed by a saline.

In case of hæmorrhage treatment will necessarily be modified by the region in which it takes place. Cuts and bruises should be cleansed and bound up, with ice, perchloride of iron, or nitrate of silver applied to the point of bleeding. In epistaxis the nasal cavities may be treated by irrigating the parts with cold water or by an absorbent-cotton plug saturated with peroxide of hydrogen; if need be, the cavities may be tightly plugged with cotton soaked in an iron solution. If the hæmorrhage arise from the socket of an extracted tooth, apply crystals of subsulphate of iron or a cotton pledget soaked in Monsel's solution, or apply caustics. Hæmorrhages from the bowel should be treated with opium to secure quiet and rest, and by cold-water injections.

Hæmophilics should be dressed warmly, should avoid cold, damp climates, and all so-called rheumatic surroundings. The joint affections may be treated much like similar conditions in chronic rheumatism, perfect rest and soothing applications being primarily indicated.

PURPURA HÆMORRHAGICA.

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UNDER the term "Purpura Hæmorrhagica" we include a clinical group of cases characterized by the association of purpura with hæmorrhages from any of the mucous membranes, less frequently into serous membranes and joints or into the substance of the viscera. First described by Werlhof in 1775, it is often known as "Werlhof's disease." It is also known as "morbus maculosus."

A careful study, however, of the cases embraced by this definition shows such a variety in their clinical course and in their etiological factors that it seems impossible to regard them even as different types of the same disease. Their symptoms, in a general way, may be alike, but in some cases they appear suddenly and peracutely without assignable cause, associated with symptoms of acute sepsis, often causing death within a few hours or days. In other cases without known cause the symptoms appear subacutely, and are less marked, the constitutional symptoms being mildly septic in character. In still others the symptoms occur either as a complication of some coexisting disease or as the result of a well-known cause. It seems better, therefore, to regard the term purpura hæmorrhagica as one purely clinical in its scope, including a number of cases distinct in their clinical course, pathology, and etiology, but which present, in common, symptoms of sufficient similarity to be included under one general name.

The study, then, of purpura hæmorrhagica is rendered more clear by dividing the cases of this disease into two groups: I. *essential*, and II. *symptomatic purpura hæmorrhagica*; the *essential* group including those cases in which the disease begins without known cause, the hæmorrhages and purpura being associated with more or less marked septic symptoms, and running a course resembling that of an infectious disease; the *symptomatic* group including those cases in which the symptoms arise from a well-known cause (as poisoning from over-use of potassium iodide), or as a complication of a severe blood or infectious disease (as in profound anæmia or in the exanthemata).

The essential cases seem to the author to constitute the only true group to which the term purpura hæmorrhagica should be rightly applied, and these will therefore be described more fully than the symptomatic cases, which should more properly be classed among the symptoms of the diseases which they complicate.

I. ESSENTIAL PURPURA HÆMORRHAGICA.

This form occurs both *subacutely* and *acutely*, the former being far the more common, and about which we know most.

SUBACUTE PURPURA HÆMORRHAGICA.

This variety of the disease is seen more frequently in females than in males. While no age is exempt, it usually attacks children and young adults. Food deficient in quantity and quality, poor hygiene, and a weak, sickly constitution predispose to the disease, but not as markedly so as in scurvy. Often it attacks those who are healthy, well fed, and well housed. There is rarely a family history of any hæmorrhagic disease, although in two cases in young girls under the author's observation the father of each had been subject to severe attacks of epistaxis in early life. The subacute cases occur in two clinical forms: (1) *ordinary cases*, and (2) *cases of Henoch's disease*.

ORDINARY SUBACUTE CASES.—This form usually begins with prodromal symptoms, anorexia, malaise, chilly feeling, and irregular rise in temperature, especially at night. These may precede the onset by several days or even weeks. In other cases there is no prodromal period. When the disease is fairly developed we have both hæmorrhagic and constitutional symptoms.

Symptoms.—*Hæmorrhagic Symptoms.*—There appear purpuric spots, usually first noticed on the extremities, though they may be generally distributed. Their size varies from that of a pinhead to that of the palm of the hand. In severe cases we may have large areas of ecchymoses, which may be extensive enough to cause gangrene of the skin. Successive crops of purpura appear during the disease, and they may be often produced by rubbing or scratching the skin. Rarely we have associated with the purpura and ecchymoses hæmorrhagic vesicles and bullæ.

There are free hæmorrhages from any of the mucous membranes—nose, mouth, gums, bronchi, stomach, intestines, and pelvis of the kidney. There may be also metrorrhagia. The most frequent sources of hæmorrhage are from the nose, pelvis of the kidney, intestines, and uterus respectively.

These hæmorrhages occur spontaneously, and not from traumatism alone, as is the case in hæmophilia. They may be moderate in their severity or profuse enough to cause the death of the patient.

Pain and swelling of the joints, especially those of the hands, feet and knees, are frequently noticed. The symptoms are identical with those of purpura rheumatica. There may be swelling of the fibro-serous tissues about the joint, or the joint-cavity may be filled by an effusion either serous or fibrino-serous. In severe cases the joint may become ankylosed or an arthritis may be caused. The primary symptoms are due to hæmorrhages either into or around the joints.

Internal hæmorrhage may occur at any time and into the substance of any of the viscera, especially the brain and its membranes, the suprarenal capsules, or the lung. These internal hæmorrhages, however, are rare in the subacute form, though more common in acute cases.

The gums may be normal or swollen, although this is denied by many writers. They may be covered by blackish scabs, and may bleed even when they are not swollen. The teeth, however, are not loosened as in scurvy.

In no case are ulcers of the intestine, due to submucous hæmorrhages, ever seen. Free hæmorrhage from the skin does not occur. Although the kidneys are frequently the source of hæmorrhage, nephritis has not been observed.

Constitutional Symptoms.—These appear in varying intensity, and are due both to the anæmia from the hæmorrhage and also to moderate sepsis. A distinct chill at the onset is rare, but chilly feelings are common and may continue through the attack. The temperature varies from 100° to 103°, or even 104°, being higher in severe cases and in children. It is higher at night. After the

severity of the attack is over the temperature gradually returns to normal: a sudden fall in temperature, with a subsequent rapid rise, is noted in cases of sudden severe hæmorrhage, especially if such occur into the viscera.

The pulse is of low tension, and somewhat rapid. It may become rapid, small, and weak. Attacks of syncope are common.

General anæmic symptoms are always present, even in cases in which the hæmorrhages are slight, but they are more severe when the hæmorrhages are profuse. They appear early in the attack and continue throughout its duration; after the attack subsides the recovery is long and tedious, and often it takes weeks or months before the blood returns to its normal condition.

Examination of the blood during the attack shows rapid diminution of the number of red blood-corpuscles, and a corresponding diminution in the amount of hæmoglobin. The white cells are at first increased in number, as is the case after acute hæmorrhage, but later their number steadily diminishes, even during early convalescence, while the number of red corpuscles and the amount of hæmoglobin are steadily increasing.

These points are well shown by the records of blood-examinations made in a case reported by Osler:

	Number of red cells.	Number of white cells.	Per cent. of hæmoglobin.
1st day,	5,350,000 (107%)	8,000	95
2d day,	3,000,000 (60%)	12,500	50
8th day,	2,500,000 (50%)	12,500	37
14th day,	3,000,000 (60%)	7,000	47
50th day,	4,000,000 (80%)	2,500	62
70th day,	4,250,000 (82½%)	. .	72

Prostration is a prominent symptom, and is always more marked than can be accounted for by the hæmorrhage and constitutional symptoms. It remains usually for some weeks after all other symptoms have disappeared. In severe or long-continued cases it may be so profound that the patient passes into the "typhoidal condition," with rapid and feeble pulse, dry brown tongue, stupor alternating with mild delirium, or even coma and death.

The spleen and liver are usually enlarged during the attack. The enlargement of the liver in some cases is well marked, and may be distinctly appreciable for weeks or months after the subsidence of the disease. The congestion and enlargement of the liver often cause a mild catarrhal jaundice, which, added to the anæmic appearance of the patient, gives a bright fawn-yellow color to the skin.

The duration of the attack varies from a few days to several weeks, but the disease may be protracted for weeks, months, or years by the appearance of similar attacks (or "relapses" of some authors). These attacks may recur at regular or irregular intervals, their usual number being four or five. In one unique case under the author's observation the attacks have persisted for fifteen years, the patient showing no signs of improvement at the end of this time. The next case of longest duration is one reported by Hryntschak, in which the attacks lasted for seven years.

Nature and Pathology.—For the blood to escape from its vessels and cause hæmorrhage we must naturally conclude that the vessel-wall must first rupture. As this does not normally occur, except from traumatism, we must also conclude that its wall is weakened either from inflammation or from degeneration due to disease, to poor blood-supply, to toxic blood, or to thrombi.

Much light has been thrown on this subject by Silberman, who gave fifteen

dogs small steady doses of pyrogallic acid until there appeared areas of stasis in the small arteries, capillaries, and veins. After pressing out the stasis-blood he injected fibrin ferment into the arteries. The dogs had abdominal tenderness, purpura, bloody vomiting, and bloody stools. Autopsy showed in the hæmorrhagic areas thrombi in the small arteries and veins, whose walls had undergone hyaline degeneration with areas of necrosis, thus allowing the free escape of blood.

Many attempts have been made to discover a specific bacterium, but before the time of Letzerich the examinations were so incomplete as to be entirely without value. Letzerich, however, in 1889 made scientific bacterial examinations, and discovered a bacillus which he believes to be the specific germ of the disease. Although his experiments have not been corroborated by others, their success still remains of the greatest value. His patient was a girl suffering from the subacute form. Bacterial examinations, scientifically performed in every detail, showed in the purpuric spots the presence of long bacilli capable of growth in gelatin, the pure cultures of which, injected into the abdomen of rabbits, reproduced the original clinical symptoms in all of the twelve cases, and in these a bacillus was found identical with that in the pure culture injected. An examination of the purpuric spots in the rabbits showed dilatation of the capillaries, emigration of white cells, and rupture of the capillary wall, permitting the escape of red cells. The capillaries were filled with the bacilli with abundant spore-growth. (The bacilli and spores had been previously described by Petrone, in his examinations of a case of Werlhof's disease, but he considered the disease to be due to a mixed infection.)

Upon squeezing the section Letzerich found that little plugs resembling hyaline casts containing bacilli emerged from the capillaries, and these he considered the result of the action of the bacillus in its products upon the fibrinoplastic elements of the blood. The liver in the rabbits was regularly enlarged, and the portal capillaries were almost occluded by an extraordinary growth of the bacilli. Letzerich considers the liver to be the breeding-place of the bacilli, the liver being to this disease what the spleen is to malarial fever. If he be correct in his conclusions, it explains both the scattering of the lesions—a bacterial embolism of the capillaries causing hyaline thrombi within them with rupture of the capillary wall—and also the tendency of the disease to relapse. While conducting his experiments Letzerich was himself seriously attacked by this disease, attributing his infection to handling his cigar while at work. This case of infection seems to prove the advisability of disinfection after an attack.

Prognosis.—This is generally good, almost all patients recovering from the primary and secondary attacks. Recovery, however, is slow, the anæmia and prostration often lasting for months after the disappearance of other symptoms. The occurrence of the secondary attacks cannot be foretold. In rarer cases the disease terminates fatally, the cause of death being either profound anæmia, fatty degeneration of the heart, with or without dilatation, from long-continued anæmia, visceral hæmorrhages, or exhaustion.

Treatment.—This is unsatisfactory, both in shortening and mitigating the attack and in the prevention of subsequent relapses, as there is no specific known that acts in this disease as quinine does in malarial fever. Our treatment, then, must be entirely symptomatic, and consists in treatment during the attack and prophylactic treatment destined to prevent future attacks.

The treatment during the attack consists in efforts to check the hæmorrhage and in the relief of constitutional symptoms. To check the hæmorrhages no one drug is certain. We employ, in turn, a number, until we find

one that is efficacious, but we may run through the entire list of hæmostatics without result. The drugs which are most frequently used are aromatic sulphuric acid, ergot, turpentine, digitalis, quinine, and gallic acid. During a hæmorrhage the patient must be kept absolutely quiet, even if morphine be required for this purpose. In all cases and at all times care should be taken to guard against traumatism, over-exertion, and excitement. Alcohol and highly-seasoned food may also give rise to a hæmorrhage. Epistaxis may be checked by astringent sprays or by plugging the nares. Uterine hæmorrhage should be treated by firm tamponage.

If the joints be affected, salicylic acid is often of service. The pain may be relieved by anodyne applications, as lead-and-opium wash, ichthyol or iodine ointment, or by the application of heat and cold. Firm compression is often grateful.

Constitutional symptoms are treated on general principles. The patient must be put to bed and on a low diet during the attack. Later he may be about the room, and a more generous diet may be allowed, vegetables and vegetable acids and fruit being especially indicated. In all cases the patient should be kept quiet and free from excitement or exertion. The bowels must be kept open, and any digestive errors corrected. Should the pulse become rapid and feeble, cardiac stimulants are indicated, especially digitalis and strychnine. Alcohol in large doses should not be used.

During the close of the attack tonics are to be given, quinine, strychnine, and arsenic being the best combination. Iron is contraindicated, as, by experience, we know that its early administration may bring on a fresh attack.

If the anæmia be marked during the attack, arsenic is the drug most efficient. It is to be given in increasing doses to the point of tolerance, then stopping its use for a day or so, and then increasing its dose as before.

If symptoms of sudden profound anæmia occur, we apply warmth to the body, hot applications over the heart, and give cardiac tonics, especially opium in small, repeated doses. Inhalation of pure oxygen gas is of the greatest service. In severe cases we employ, in addition, rectal or hypodermatic injections of a warm sterilized saline solution. Several pints can be given in this way with great improvement of the symptoms, although this may be but temporary. Arterial transfusion is not to be used, because of the danger of traumatic hæmorrhage. Elevation of the foot of the bed and ligatures applied to the extremities are often followed by good results.

The prophylactic treatment employed during and after convalescence is intended to lessen the chances of subsequent attacks.

The patient must live and work in airy, sunny rooms and take graded exercise in the open air, for fresh air and moderate exercise are of the first importance. The plumbing must be in perfect sanitary condition. The diet should be wholesome and varied, and every digestive error corrected.

For the anæmia, arsenic in small continued doses is by far the best treatment. It should be continued until the blood becomes normal. It may be combined with quinine and strychnine. Iron is not to be used at first, but several weeks after the primary attack has subsided it should be given in small doses at first, then slowly increasing. Should a relapse threaten, the iron must at once be stopped.

Prostration is to be treated on general principles by rest, fresh air, graded exercise, and change of climate. The climate most suitable is one in which the air is light, dry, and bracing; and the location must be inland, as we find that the disease more extensively prevails on the sea-coast.

As the disease is probably due to an infectious specific germ, and as the sub-

sequent attacks are also probably due to reinfection, it seems certainly better to disinfect the room and the clothes of the patient after the illness. The more we study this disease, the more we incline toward such disinfection.

HENOCH'S DISEASE.—The severe form of the subacute cases was first described by Henoch, and is known as "Henoch's disease" or "Henoch's purpura hæmorrhagica." This form occurs with greater frequency in children, especially between the ninth and twelfth years. It has been observed, however, between the third and forty-sixth years. It occurs five times more frequently in males than in females. It is a rather rare form.

Symptoms.—There is usually a short prodromal period with malaise, slight fever, and sometimes with pains in the joints.

The onset is manifested by the appearance of purpura, in severe cases accompanied by ecchymoses, these differing in no way from those described under the first form. Immediately after the purpura, appear the severe abdominal symptoms which characterize the disease. There is marked pain and tenderness over the abdomen, the pain being of a colicky character, with exacerbations of great intensity. The abdomen is rigid and retracted. There is severe rectal tenesmus with bloody stools and severe vomiting, the vomited matter being either like that of acute gastritis or containing blood. These abdominal symptoms seem to be due to submucous hæmorrhages or to hæmorrhagic infarctions caused by thrombi in the small blood-vessels of the gastro-intestinal wall, which become degenerated and rupture, allowing free hæmorrhage. Patches of intestinal ulceration result in rare cases, and rupture into the peritoneal cavity with fatal peritonitis may occur even after apparent recovery.

These symptoms continue with great intensity for one or two days, and then gradually subside. They may continue longer, but in such cases there are periods of temporary improvement. Joint symptoms may appear as in the first form. Hæmaturia is seen in one-fifth of the cases. The spleen is usually enlarged, and there is a slight rise of temperature during the attack.

After such an attack the patient is liable to have a series of similar ones, usually at short intervals. There are generally four or five such, but their number has been recorded as high as twenty.

The nature of the disease is unknown. No specific micro-organism has as yet been found, but as the reported cases are few, it is possible that in time one will be discovered, either Letzerich's bacillus or some other bacterium producing the same results.

The duration varies according to the length of the attacks, their number, and the intervals between them. It is usually six to twelve weeks, but may be limited to a week or be extended to nine months.

Prognosis.—This is fairly good, being better in children (mortality, 5 per cent.) than in adults (mortality, 25 per cent.). The possibility of intestinal rupture and peritonitis, though rare, must be taken into account.

Treatment during an attack is purely symptomatic. Between the attacks we improve the general condition in every way.

ACUTE PURPURA HÆMORRHAGICA

is far more rare than the subacute form. The same symptoms are present, but run an acute and more severe course, overwhelming the patient by their violence and the rapidity of their onset. The acute form differs, moreover, from the subacute in the severity of septic symptoms, in the frequency of visceral hæmorrhages, and its disposition to attack pregnant women. We can

broadly subdivide the acute cases into three groups: (1) cases with marked sepsis; (2) cases with visceral hæmorrhages; (3) cases complicating pregnancy.

1. CASES WITH MARKED SEPSIS.—These present both severe hæmorrhagic and septic symptoms, but the latter are so predominant that the course of the disease is essentially that of acute septicæmia.

The attack usually begins by a chill or chilly feelings, with a rise in temperature to 103° or 104° F. Hæmorrhagic symptoms soon develop, purpura and hæmorrhages from any of the mucous membranes. These are severe, and are not readily controlled by treatment. Septic symptoms are marked from the onset—severe prostration, mental apathy, stupor, or semi-coma, alternating with periods of restlessness, anxiety, and mild delirium, and finally, in fatal cases, complete coma. The temperature remains high, 103° to 104° , but in severe cases it may rise to 105° or 106° . The pulse becomes rapid, feeble, and irregular; and the patient usually dies early in the disease, either from sepsis or from acute anæmia.

The following case, personally observed, illustrates most typically the clinical course of this form:

L. M.—, female, nineteen years, had always lived in most affluent circumstances; had never been sick except from slight anæmia for the past two years. Father when a boy would bleed severely from slight causes. No further hæmophilic history.

March 7th, 1 A. M., slight chill without rise in temperature. Very nervous and anxious. 12.45 P. M., marked chill, fever rising to 103.5° , and epistaxis becoming more and more profuse in spite of every effort to check it.

March 8th, 1 P. M., first seen by author. T. 98.4° ; P. 130, irregular and weak; marked pallor of skin; prostration profound; complete mental apathy, though her mind was clear when she was aroused. New purpuric spots appearing. Gums normal. No evidence of endocarditis nor of any other appreciable disease. Spleen enlarged; epistaxis still continuing, the blood being dark and not coagulating. Profuse uterine hæmorrhage. Hæmorrhages were checked by plugging posterior and anterior nares with cotton dipped in collodion and by firm tamponing. 8 P. M., T. 102.8° ; P. 130–180, weak and irregular; semi-coma, alternating with periods of restlessness and mild delirium. Still slight hæmorrhages from nose and uterus in spite of former treatment. 10 P. M., about a pint of warm sterilized saline solution was given by rectum and by hypodermatic injections, with slight but temporary improvement. Cardiac tonics, whiskey, and digitalis were freely administered.

March 9th, 9 A. M., T. 104.8° ; P. 148; R. 32. Large offensive tarry stool of altered blood. Injection of saline solution continued. 6 P. M., complete coma. T. 106.2° ; pulse weaker and flickering.

March 10th, 2 A. M., she died, two and a half days after the onset of the disease. No autopsy was permitted, and bacterial examinations could not then be made.

Etiology.—There is no known cause for this disease. It occurs more frequently in men than in women. The average age of the males affected is twenty-eight years; of the females, twelve years. It has been observed, however, between one and seventy years of age.

The average duration of the attack is about one week, although it may last from one to twenty days.

Prognosis.—The prognosis is bad, 75 per cent. of the cases terminating fatally.

Treatment consists in—(1) checking the hæmorrhages by plugging the nares, by firm tamponage, or by the use of hæmostatics, as described in the sub-acute form. (2) In controlling the sepsis. This is often more than we can do, although in some cases alcohol in large doses seems to do good. (3) In the treatment of dangerous symptoms. Heart-failure is to be treated by hot applications over the precordium and by cardiac stimulants. The restlessness and anxiety are best controlled by opium given in small doses. Profound anæmia

is to be treated by external warmth, rectal and hypodermatic injections of a warm sterilized saline solution, elevation of foot of the bed, and ligatures applied to the extremities. Arterial transfusion is contraindicated.

2. CASES WITH VISCERAL HÆMORRHAGE.—In these cases the brain and the suprarenal capsules are the organs most frequently involved.

In the brain cases the disease begins with the ordinary symptoms of acute purpura hæmorrhagica. After several days these are followed by those of meningeal or cerebral hæmorrhage, usually multiple, and without any especial seat of selection. It is seen far more frequently in males than in females.

Illustrative Cases :

1. Girl, aged two years. For two days diarrhœa and vomiting; then purpura, fever, and collapse. Death in a few hours from multiple hæmorrhages into the medulla. (Züelchauer, *Berl. klin. Wochensch.*, 1869, No. 17.)

2. Young man. General acute symptoms. Death on fourth day from hæmorrhages into left Sylvian fossa, pons, and ventricles. (Kurkowski, *V. und H. Jahresbericht*, 1885, ii. p. 493.)

In cases of hæmorrhage into the adrenals the course of the disease is exceedingly acute, and death results in a few hours after the onset.

Illustrative Cases :

1. Soldier, aged twenty-two. Purpura; hæmorrhage from mouth, lungs, and kidneys. Death in seven hours from adrenal hæmorrhage. (Bourrieff, *V. und H. Jahresber.*, 1878, ii. p. 275.)

2. Male, aged two years and nine months. Purpura, fever, and collapse. Death in fifteen hours from adrenal hæmorrhage. (Wolff, *Berl. klin. Wochensch.*, 1879, No. 18.)

3. CASES COMPLICATING PREGNANCY.—In the cases in which the disease attacks pregnant women we have the ordinary acute symptoms at first, followed by miscarriage and post-partum hæmorrhage. It may also follow labor at term. The disease runs a rapid course, and recovery is rare.

Illustrative Cases :

1. Female, aged twenty-one, six months pregnant. Purpura four days; then rapid onset of increasing purpura, with hæmorrhages from nose, gums, kidneys, and stomach. Miscarriage sixth day, with post-partum hæmorrhage. Death on eighth day, four days after the acute onset. (Puech, *Annales de Gynécologie*, xvi., 1887, p. 273.)

2. Female, aged thirty. Five previous normal labors. Seven months pregnant. Purpura, with miscarriage in a few hours with post-partum hæmorrhage. Death on second day. (Phillips, *Brit. Med. Journal*, Nov. 13, 1886.)

3. Female, aged thirty-two. Seven previous normal labors. Seven months pregnant. Purpura, hæmorrhages from nose and mouth. Miscarriage on third day, with placental hæmorrhage. Recovery in two weeks. (Phillips, *loc. cit.*)

When we study these acute cases together, we are struck with their similarity to the class of acute infectious diseases. The absence of assignable cause, the rapidity of the onset, the multiplicity and scattering of the lesions, the enlargement of the liver and spleen, and the constitutional symptoms out of proportion to the lesions, seem to prove by analogy the assertion that we are dealing with an acute infection, the nature of which is at present unknown. Comparing these cases, however, with those of the subacute form, the identical symptoms are found in each, and it seems most probable that in both forms we are dealing with the same disease in all essential features, differing only in the intensity and rapidity of the infection. As the infection in the subacute cases seems to be due to the presence of Letzerich's bacillus, it is more than possible

that the acute cases may be due to a more intense infection by the same germ. Much attention has been called to the relationship of essential purpura hæmorrhagica to two diseases of the hæmorrhagic group—purpura simplex and purpura rheumatica.

Purpura simplex is due to a variety of causes. In some cases the cause is apparent, as in severe anæmia, debility, after certain drugs, or occurring in infectious diseases. In other cases no cause can be found and the nature of the disease is obscure. In either we may have mild or severe constitutional symptoms.

In purpura rheumatica we have not only simple purpura, but also pain and swelling of the joints. Formerly it was regarded as a separate disease from purpura simplex, but of late efforts have been made to associate them, purpura rheumatica being considered either as a purpura occurring in rheumatic subjects, thus accounting for the joint symptoms, or as a severe purpura simplex, in which hæmorrhages occur in and around the joints. The author regards the latter supposition as the more correct, as in all hæmorrhagic diseases, purpura hæmorrhagica, as well as scurvy, multiple sarcoma, etc., the joints may be affected, together with the appearance of purpuric spots. If this view be correct, why regard them as separate diseases? Is it not justifiable to consider purpura rheumatica as an intenser form of purpura simplex with hæmorrhagic joint lesions?

If purpura hæmorrhagica be due to an infection, may not the cases of purpura simplex occurring without known cause, and cases of purpura rheumatica not associated with rheumatism, be considered as lighter forms of the same infection, especially as in some cases of subacute purpura hæmorrhagica, purpura or purpura with joint symptoms may be the most marked features, the free hæmorrhages being of very slight importance, often not appearing for several days after the other symptoms? Even in the acute form is this seen, as the case of Puech's, cited on the preceding page, illustrates, the purpura alone existing four days before the onset of acute symptoms.

In support of this theory may be cited cases of secondary purpura hæmorrhagica, such as those occurring after the administration of certain drugs, in which small doses in some patients produce merely purpura, while large doses cause, in addition, free hæmorrhages and marked constitutional symptoms. The only difference seems to be that in one case we are dealing with a cause unknown, though probably bacterial, while in the other the cause is known, and by its intensity we have all grades, from simple purpura to purpura hæmorrhagica, even of an acute type.

II. SECONDARY PURPURA HÆMORRHAGICA.

Under this class we include those cases of purpura and free hæmorrhages which complicate some existing disease or to which a definite cause can be assigned. In nearly all of these cases we may have either a simple purpura or purpura hæmorrhagica with constitutional symptoms of a mild or severe character, in some even running a fatal course. Only a brief mention can be made of these cases.

(1) *Cases due to the Administration of Certain Drugs*, potassium iodide, chloral, quinine, and salicylic acid being the ordinary drugs causing such a result. There is a great difference in their action in different patients, some developing no symptoms, others a simple purpura, while in still others we have a striking exhibition of spreading purpura, free and internal hæmorrhages, with coma, collapse, and even death. These various types can proceed from the

same cause acting more intensely upon some patients than upon others, either from a maximum of cause on the one hand or the minimum of personal resistance on the other.

(2) *Cases which Accompany or closely Follow Severe Infectious Diseases*, such as acute atrophy of the liver, snake-bites, typhoid fever, pneumonia, and the exanthemata ("black measles," etc.). In these cases we have various grades, from simple purpura up to acute purpura hæmorrhagica. Many authors attribute such a complication to an added infection of essential purpura hæmorrhagica complicating the primary disease. Henoch, for example, reports a case of a child with lobar pneumonia in whom a supposed infection of purpura hæmorrhagica occurred two days after crisis, causing death from collapse in twenty-four hours. If a drug like potassium iodide will so disorganize the blood or render pervious the blood-vessels, why may not the poison of an infectious disease produce the same result without supposing an added infection of a new disease? It is no argument against this view that purpura hæmorrhagica may appear after the crisis, because we know that a temperature crisis does not mark the end of the disease, but only, as Fraenkel has recently demonstrated in pneumonia, the end of the fever-producing quality of the infecting germ.

(3) *Cases of Severe Jaundice* may be accompanied by purpura and hæmorrhages. These seem to be due to the disorganization of the blood from the cholæmia.

(4) *Cases of Profound Anæmia, Leukæmia, or Pseudo-leukæmia, and of Exhausted and Cachectic Conditions.*—In these we may have simple purpura, purpura hæmorrhagica, or continued hæmorrhage after operations or injuries. We do not know whether to attribute these hæmorrhages to blood-changes or to changes in the wall of the small arteries.

(5) *Cases of New-born Infants with Congenital Syphilitic Changes in the Arterial Walls*, producing purpura, bloody sweating, and free hæmorrhages, especially from the umbilicus.

(6) *Cases of New-born Infants without Syphilitic Parentage.*—This form, according to Partridge, occurs in about 1 per cent. of cases, with a mortality of 60 to 75 per cent. He attributes its causes to the change of functional activities and to the altered circulation, allowing a brief interruption of the nutrition of the vessel-walls sufficient for the transudation of their contents.

(7) *Cases complicating Malignant Endocarditis*, the purpura and hæmorrhages being probably due to embolism of the capillaries by vegetation-fragments, and their subsequent degeneration and rupture.

(8) *Cases of Multiple Sarcomata, with Purpura*, with free hæmorrhages, purpura, rheumatic pains, and fever. It is hard to say whether these result from malignant cachexia, with blood-changes, or from emboli of sarcomatous fragments lodging in the small blood-vessels, causing their degeneration and rupture.

(9) *Cases occurring after Fright, Deep Emotion, Hysteria, and Hypnotism.* In these cases the hæmorrhages seem to be due to vaso-motor relaxation or to enfeeblement of the arterial walls sufficient to allow of the escape of their contents. This latter explanation is warmly endorsed by Weir Mitchell.

SCORBUTUS.

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INFANTILE SCURVY is a constitutional disease produced by improper feeding, characterized by swelling, disability, excessive tenderness and pain on motion in the lower extremities, and spongy gums: it is further characterized by rapid recovery under corrected regimen.

The first case of infantile scurvy was reported by Jalland, and the report summarized in *Virchow's Jahresbericht* for 1873, but England has been the source of most of the reported cases and most of the literature of scurvy in children. To W. B. Cheadle and Thomas Barlow of Great Ormond Street Hospital is due the credit of "having first shown on clinical grounds the true affinities of this form of infantile cachexia" (Cheadle), and of demonstrating the anatomical nature of the disease from post-mortem examinations (Barlow). Prior to the work of these observers infantile scurvy had been regularly regarded as acute rickets or gone astray as purpura hæmorrhagica.

The first case of infantile scurvy in the United States was met with upon the autopsy table of the New York Foundling Hospital. A second was soon afterward recognized in consultation, treated, and recovered. At the time of the publication of the first edition of this work 11 cases were on record, and were made the basis of the first article on infantile scurvy in an American textbook. In 1894, 36 cases were collected and reported by Crandall and Northrup. Since that time cases have been reported from all parts of the country. One observer alone (Rotch of Boston) has seen 60 or 70 cases. The subject of infantile scurvy can, therefore, rightly claim the attention of the general practitioner.

Etiology.—The cause of scurvy in children is persistent feeding with improper foods. Examining the dietaries of scorbutic children, we find enumerated almost everything that could possibly be employed as food for a child—all manner of proprietary foods, condensed milk, porridge, oatmeal and barley-water, various mixtures of cow's milk and cream. They agree only in one respect: they all lack or have been deprived of the quality which we designate as fresh or "live."

When milk or cream has been given it has regularly been deprived of this quality by sterilization. For some time there has been active debate whether prolonged sterilization of the food could of itself cause scurvy. The writer has recently seen 2 cases of typical scurvy developed in children fed upon perfectly proper milk mixtures which had been sterilized by prolonged boiling. Both recovered promptly on the same food when the over-sterilization was stopped: moreover, one of them has now gone for several months upon the same food unsterilized, without any return of the symptoms. Like experience has been recorded by Starr and Holt.

Scurvy among nursing infants is very rare. In the first case met with

in this country the child had been nursed by a woman who suckled her own child as well. The latter thrived; the foster-child developed scurvy. There is little doubt in such a case that the child was starved into scurvy.

Southgate has also recorded a case of scurvy in a nursling. Moreover, the analysis of the milk made in this case shows it to have been rich in quality. In the light of all other observations we can only say that this case stands unique and unexplainable.

The patent baby-foods are, by all means, the most frequent offenders in the production of scurvy. The measure of the responsibility of any particular one seems to rest only on the extent of its popularity. Those most widely used are most often met with in the scurvy records.

As the number of reported cases increases the stronger becomes the indictment against the patent foods. There seems no greater surviving fallacy in medical practice than the routine feeding of infants with patent products of commercial firms. Condensed milk deserves to rank with the other proprietary foods.

Surroundings seem to have little influence upon the production of this cachexia. Most of the reported cases have been observed in private practice. In the great majority the surroundings have been good, in many luxurious. The affection has been met with in all parts of the land, both in cities and in the country, along the seaboard and on the mountains of Montana. Persistent feeding with improper food can produce scurvy anywhere. The disease is usually met with after the sixth month and under two years, but these limits cannot be regarded as absolute. It takes time to develop scurvy, no matter how bad the diet, and after the second year the diet usually becomes so general that all danger is removed.

We are still unable to reach the ultimate cause of scurvy. It seems unquestionably to be deprivation, but of what has not yet been determined. All that we can say is that the missing elements are found in fresh milk and fresh fruit-juice.

Pathology.—The lesions of infantile scurvy are well set forth in Northrup's report of the autopsy on his first case. The child was emaciated, its eyelids swollen and ecchymotic. The gums were prominent, spongy, dark, covered with dried blood, the lips blood-stained. The pale, thin face, with two black eyes, gave a most striking appearance to the dead baby. The main interest lies in the condition of the legs. Left thigh symmetrically enlarged, larger than the right, although both were obviously above normal in size. Left femur was normal at its upper extremity, epiphysis, and end of shaft. The lower half was invested by a black, grumous, subperiosteal layer of blood two or three millimetres thick. The lower epiphysis was detached; the lower end of the shaft macerated, eroded, and soft, lying loose in the black, disintegrating blood-clot. The femur of the right leg was surrounded for its lower two-thirds by a thinner, black, subperiosteal blood-layer. The lower epiphysis was not detached, though both it and the shaft were congested. No hæmorrhage into joints. The right and left tibiæ were surrounded by a thin, dark, hæmorrhagic layer beneath the periosteum, and the proximal portions of both were congested. The fibulæ and bones of the upper extremity were normal. Microscopical examination of the bone disclosed no syphilitic or rachitic changes, and no inflammatory changes in bone or periosteum. The softened, macerated bone gave no evidence of suppuration, but there was moderate congestion of the fellow-femur and upper extremities of the tibiæ. A small amount of blood, dark and disintegrated, was found in the intestines; no lesion discovered. The accom-

panying illustration (Fig. 1) was drawn from a specimen which consists of a lateral half of the side less affected.

To this we need only add that subperiosteal hæmorrhages may occur upon any of the bones—those of the upper extremity, of the cranium, of the thorax. There may also be hæmorrhages from various mucous membranes—the nose, the stomach, bladder, etc.

FIG. 1.



Symptoms.—The characteristic symptoms of infantile scurvy are the swollen, spongy, purple, and easily bleeding gums, and extreme pain on motion, tenderness, swelling, and disability in one or both lower extremities. Examination of the affected extremities reveals a fusiform or cylindrical swelling about the long bones. The affection is usually most marked about the femur, but the bones of the leg or ankle may be involved. The affection is usually bilateral, but not symmetrical, one extremity presenting more marked changes than the other. In a few cases the upper extremities have been involved, but these cases are rare, and in almost all thus far reported there was an antecedent affection of the legs. The joints themselves are not involved in the process. The affected limbs are usually held in a semi-flexed position (Fig. 2), and no attempt is made to use them, so that the disease is often mistaken for a paralysis. This disability is spoken of as pseudo-paralysis. "Rheumatism of the legs" is another favorite diagnosis for this scorbutic affection of the extremities, but, as already noted, the joints themselves are not involved in infantile scurvy. The pathological lesions already described render these symptoms readily explicable.

In addition to the characteristic symptoms, subcutaneous ecchymoses or hæmorrhages are of frequent occurrence. They may be seen upon any part of the body, but are especially characteristic about the orbit, giving the little patient a typical "black eye."

Hæmorrhages may also occur from mucous membranes other than the gums, so that there may be bleeding from the nose, stomach, intestine, or bladder; but such hæmorrhages are seen only in the severer types of the cachexia.

For weeks before the development of the evidences of scurvy the child may suffer from gastric or intestinal disturbances, with vomiting, colic, diarrhœa, or constipation. In the severer cases a sallow, muddy complexion, due to severe anæmia, is often met with. The examination of the blood shows the changes of simple anæmia. Many of the cases are marantic, but scurvy may also be seen in children who have suffered from no gastric or intestinal disorder and are well nourished and ruddy.

The affection of the gums is seen only about the teeth. If the child has no teeth, the gums will appear normal. In the report of Crandall and

Specimen from a case of Infantile Scurvy, showing subperiosteal hæmorrhage about femur and tibia of the side less affected. (Drawn from the specimen preserved in the Museum of the College of Physicians and Surgeons, N. Y.)

Northrup the condition of the gums was noted in 32 cases. Of these, 2 had no teeth; the gums were normal. Of the remaining 30, 24 had what was termed "spongy" gums, 3 had ulcerated gums, in 3 they were described as "bleeding." In 34 other cases of which the records are available, 31 had spongy gums; in 3 the gums were normal. Of the latter, 2 had no teeth. One, although it had two teeth and presented a typical scorbutic affection of the extremities and subcutaneous ecchymoses, had no mouth-symptoms whatever. The affection of the gums, although regularly present, cannot, therefore, be considered essential to the diagnosis.

The constitutional disturbance of scurvy may be of any degree of severity, depending upon the gravity of the affection and the time of observation. In the mildest cases the baby may appear perfectly well, except for the pain on motion of the extremities. In the severer types there are marked anæmia, emaciation, fever, and prostration, which may result in death. Fever, if

FIG. 2.



Infantile Scurvy : characteristic attitude of the legs.

present, is usually slight, but may reach 102° or 103° F. It is apparently dependent upon accompanying disturbances, and not upon the scurvy itself.

RELATION TO RICKETS.—The relation of scurvy to rickets has long been the subject of debate. Previous to the work of Barlow and Cheadle infantile scurvy was regularly described as "acute rickets," and in the early days of observation rickets was supposed to constantly precede or accompany the appearance of scurvy. In the report previously quoted rickets was referred to nineteen times. Five cases showed marked rickets, 6 slight; in 8 it was definitely not present. In 34 other cases, of which the records are available, rachitis was noted as present only in 5, and in most of these the only evidence of rachitis was "beaded" ribs. Rickets and scurvy are both developed during infancy. Improper diet is a causative factor in both, but either may be developed without the other. The lesions of rickets are found in the bones; those of scurvy are evidently in the blood-vessels. The subperiosteal or subcutaneous hæmorrhages of scurvy may be promptly absorbed and the child left perfectly well. The changes of rickets are more or less permanent. The correction of diet sufficient to cure scurvy in a few days makes no impression

upon rickets. The two affections may be comrades; they are not generically related.

Illustrative Cases.—Three cases representing the several types of infantile scurvy will be presented:

CASE I.—A mild case. A boy, aged twelve months; only child, birth normal. Parents both very well and surroundings good. Child artificially fed from birth. For first two months he was given diluted condensed milk. Thereafter the food consisted of a mixture of cow's milk regularly boiled for fifty minutes. Upon this the child thrived, became fat, rosy, and vigorous. He had no gastric or intestinal disturbance. The bowels moved twice a day; the passages were quite normal. In short, he had been considered a remarkably healthy and vigorous baby until he was ten months old. His mother then noticed that he would no longer attempt to stand or use his legs in any way. At the same time he began to cry whenever moved or touched, and sweated a great deal. He became more and more fretful, and cried a great deal, especially at night. These symptoms persisted and became worse up to the time he was brought to the clinic of the New York Orthopedic Dispensary.

Examination.—A large, well-nourished baby, of good color, and with no evidence of rachitis. Lying perfectly quiet in his mother's lap, he would smile and play as though perfectly well, but the first suggestion of a touch or any motion called forth piteous wails. The four incisors were present. About the upper pair the gums were purple, the mouth otherwise normal. Both lower extremities were swollen from the ankle to the knee. Although the least attempt at examination made him scream with pain, he made no effort to withdraw the legs or move them in any way. To touch there was a sensation of deep thickening about the long bones of the legs. In every other way the child was perfectly normal.

For treatment the mother was directed to give him the same mixture of milk unboiled, with two or three teaspoonfuls of fresh orange-juice daily. Two days after beginning this treatment the baby slept all night for the first time in two months. In five days he ceased to cry and the legs could be moved without pain. In two weeks he was able to stand, and since that time has been the "picture of health."

CASE II.—A typical case. A girl sixteen months old, a second child. At the time of the consultation the father and mother of the little patient were present; both were within the thirties, healthy and vigorous, the father looking like a hardy yachtsman. The family history on both sides was good. The home was located in the most hygienic surroundings of up-town New York. When the child was in its fourth month the mother's milk failed to be of sufficient quantity, and soon thereafter ceased altogether. One of the proprietary foods was then given. By some misunderstanding this food was diluted with water and milk, the proportion of the latter being too small. For a time the child apparently thrived very well, although it was rather backward. Its digestion was good, its bowels were reasonably satisfactory, and it seemed satisfied with its food. It never gave any evidence of rickets; teeth in normal number made their appearance at the usual time.

Three weeks before the visit spoken of (this fact was subsequently elicited after close questioning) the nurse had noticed some *change in the child's gums*. The change was not marked.

One week later the patient developed trouble *in the right lower limb*, evidenced by worrying, sensitiveness on handling, and a tendency to keep the limb nearly straight. There seemed no reason why the case should not speedily

come out of its condition of slight depression, as the food was improved and antirheumatic treatment instituted.

During the succeeding week very little is known concerning the child; the parents were absent from home; the family physician was not called; the nurse drew no conclusions from the now rapidly changing gums, and as to the "rheumatism" the progress was slow.

The child cried on seeing a strange face, becoming alarmed also for the safety of its lame leg. In the wry face of crying the little patient fairly unbuttoned from between its lips two rows of irregularly nodulated, purplish gums, from the summits of which the points of its teeth barely protruded. *In the upper spongy row was a depression with ulcerated walls and sloughing shreds. The gums were dark, and bled freely* in the act of crying from compression of the lips alone. There was nothing further abnormal about the face beyond a worried expression; no ecchymoses, no petechiæ; conjunctivæ were normal; no evidence of unhealthy condition of the mucous membrane of the nose. There was no history of nose-bleeding, no hæmaturia, no hæmorrhages from the bowels. The child was now stripped of all clothing and laid upon its back on the bed. It continued to whimper, throw its arms about freely, draw up its left leg; as for its right, it could move it slowly, but only a little, and could not be induced to flex it. *The right thigh was distinctly larger than the left* to observation; by measurement it showed a difference of about two and a half inches, which, considering the thin thighs of the small patient, augured a marked difference. The enlargement was fusiform, greatest just above the knee. Apart from the *spongy gums and swollen thigh* there were no external manifestations.

This case promptly recovered on corrected regimen.

CASE III.—Fatal scurvy; child of eighteen months; autopsy. This child was an inmate of the New York Foundling Hospital, and was what is called a "nurse-baby;" that is, she was nursed by a mother, who, in addition to her own baby, nursed a second of about equal age. Her own child thrived; the second furnished the example of malnutrition and the pathological specimen already referred to. Since we are considering a case of scurvy developing in a breast-fed (*sic*) child, it is well to bear in mind the above facts, and the added fact that nearly all babies nursing two at one woman require more or less artificial food. We are justified in forming our own conclusions as to which was nursed more and which less; we know which baby was hers and which was not, which thrived and which developed fatal scurvy.

Briefly, the history of the illness was as follows: The foster child when sixteen months old was observed to be failing, and, as the history reads, "on account of impaired nutrition was taken from the breast and was given vegetable acids." In the seventeenth month of life, which was one month before death, the right leg and knee became swollen and tender. Temperature was 101° F. After two days the symptoms seemed temporarily to disappear. Two weeks before death, and six weeks after the weaning, the child appeared to be very sick; her gums were swollen, smoky-black, and bled freely; two days later her left eyelid became swollen, black, having the appearance of the classical "black eye." Temperature thus far continued about 101° F. One week later there developed the physical and rational signs of pneumonia. At this time her other eyelid became ecchymotic and the other thigh markedly swollen.

During the remaining days of life the little patient became excessively anæmic, having a metallic pallor, which gave a particularly wretched appearance with the contrasting ecchymoses about the eyes. Her passages were

black and pasty; no petechiæ; the child failed rapidly and died with pronounced symptoms of pneumonia. (For autopsy see "Pathological Anatomy.")

Prognosis.—When recognized and properly treated scurvy disappears with almost magical rapidity. Unrecognized and improperly treated, it may readily prove fatal. The outcome depends upon the diagnosis. Under proper treatment complete recovery ought to be obtained in three weeks.

Diagnosis.—Scurvy in infants is most frequently mistaken for "rheumatism of the legs," infantile paralysis, acute rickets, or an osteomyelitis. Sarcoma of the femur and simple stomatitis have also been recorded in the category of erroneous diagnoses, and the limbs have been laid open for pus only to find pure blood.

Four points suffice for an absolute diagnosis:

1. The age of the child—over six months, under two years.
2. The history of improper feeding, especially proprietary foods, condensed milk, or milk mixtures sterilized by prolonged boiling.
3. The painful, swollen extremities, without local heat or redness, and without involvement of the joints.
4. The spongy, purple, easily-bleeding gums.

A thorough examination should establish the diagnosis in any case. If doubt remains, a few days' treatment will settle the question. On an anti-scorbutic diet improvement should be prompt.

Treatment.—Briefly, correct the diet. Put the child upon a proper mixture of cow's milk, raw or pasteurized; when possible, give milk warm from the cow. Administer orange-juice freely, a teaspoonful every two or four hours. Improvement will be almost immediate, and complete recovery not long delayed.

Stimulants will be required only in cases of extreme exhaustion. If the anæmia is severe, iron is indicated. It is best given in the form of the powder (Quevenne's iron), gr. j to ij, *t. i. d.*

As a rule, the dietetic treatment is all that is required.

PART VI.

DISEASES OF THE DIGESTIVE ORGANS.

DISEASES OF THE MOUTH AND DENTITION.

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I. DISEASES OF THE MOUTH.

THE mouth of an infant differs in many respects from that of an adult or even a child: up to the third or fourth month of life it is to be looked upon merely as a passage-way for food. Then comes the first outpouring of saliva, and with it the functions of the mouth are increased by that of incipient digestion, which reaches its full development after a period that varies in individual cases. The lack of saliva produces more or less dryness of the infant's mouth, a coating of the tongue due to epithelial cells, detritus, and food, and a peculiar glistening appearance by reflected light. After saliva is formed the child does not, at first, know what to do with it, so that, even when normal in quantity, the greater part of it is not swallowed.

For most of the inflammations of the mouth the etiology is still a matter of surmise. While there can be no doubt that lower forms of life must play a very important rôle in their production, yet as a matter of fact but few forms of stomatitis can be definitely ascribed to this cause. The mouth is a veritable culture-tube for microbes and lower forms of life, but, as a rule, they do not produce disease. General conditions of the patient must seriously be taken into consideration (syphilis, rickets, scurvy); possibly these may produce a soil favorable to low conditions of life, resulting in the production of troubles in the mouth. Local conditions within the mouth must always be sought in examining a case—lack of cleanliness, rough attempts at cleansing, sharp or diseased teeth, the introduction of irritants or poisons; while, on the other hand, causes may be found only in diseased conditions of remote organs. One important fact must always be taken into consideration, that the glands of the mouth are not only secretory, but also excretory, so that substances taken into the circulation, as well as others formed within the body, may leave the body by means of these glands and produce local lesions.

In the matter of treatment care must always be exercised in removing the cause of the disease; where this is impossible, purely symptomatic treatment is called for, and this, in the main, is antiseptic in nature. The most potent mouth-antiseptics are potassium chlorate, potassium permanganate, silver nitrate,

and sodium salicylate. Each one has its own indications, but the first and second are almost universally serviceable. Potassium chlorate, especially, when used internally, requires cautious administration on account of its effects upon the blood and the kidneys. It is safe to say, however, that the danger has been largely over-estimated by some, and in comparison with the frequency with which the drug is used the number of cases of poisoning is exceedingly small.

The examination of the mouth should be thoroughly conducted, without force, but in such a way that all parts can be seen to advantage. It is necessary to insist upon this part of clinical examination, since, simple though it be, it is frequently neglected, so that very valuable aids to diagnosis in many diseases are overlooked.

The classification which follows is one which is principally based upon clinical data; it is completely satisfactory as a working formula up to the present, but will undoubtedly require revision in the future. The term "stomatitis" is retained for many reasons, not the least important being that it has been used quite universally. The following are the forms of stomatitis: I. Stomatitis catarrhalis; II. Stomatitis aphthosa; III. Stomatitis mycosa; IV. Stomatitis ulcerosa; V. Stomatitis gangrenosa; VI. Stomatitis crouposa; Stomatitis diphtheritica; VII. Stomatitis syphilitica.

I. STOMATITIS CATARRHALIS.

Also called simple stomatitis, of which there are two kinds—local and general.

Etiology.—Two things must be taken into consideration—an irritant and the mucous membrane. In healthy children the mucous membrane resists to a greater extent than in children sick with any disease whatsoever. The most favorable conditions for the production of stomatitis catarrhalis are to be found in children with acute febrile disease and in bottle-fed babies. The irritants are either mechanical, thermal, chemical, or to be traced to some lower form of life acting mechanically or chemically. In healthy children teething does not produce stomatitis, and it is denied by many that this process is even a predisposing cause. Lack of cleanliness, over-cleanliness, and food introduced at too high a temperature are common causes for this trouble. Many of the acute infectious diseases produce stomatitis catarrhalis, which then precedes the appearance of the characteristic lesions within the mouth. Nearly all other forms of stomatitis are preceded by this form—most especially is this the case with stomatitis mycosa; and all other forms are associated with more or less catarrhal inflammation. In all probability, substances excreted by the glands of the mouth, as the result of faulty digestive processes in the intestines or of incomplete elimination, will be found to be of vast importance in the etiology. This will be the most rational way of explaining the frequent concurrent appearance of diseased processes within the mouth and the intestinal tract. For the localized form, it is a local irritation—a sharp tooth, a discharging abscess, or the rubbing of the gums to facilitate teething.

Symptoms.—We may recognize two varieties, the erythematous and the true catarrhal. In the erythematous form the whole mucous membrane of the mouth is of a deep-red color, produced by hyperæmia. The blood-vessels are sometimes subjected to such great pressure that rhexis occurs, or red corpuscles may be forced into the submucous tissues, and the hæmoglobin may there be changed to hæmatoidin, with a resulting distinct yellow discoloration. This condition is frequently found in the mouth of the new-born; erythema

of the mouth may be looked upon as normal during this period of life, requiring no treatment except gentleness, and is of no special importance.

In pertussis and the acute exanthemata there is produced a peculiar form of erythematous change. In pertussis and measles it consists of a blue tint given to the tongue and the buccal cavity; in scarlatina the whole mouth is more or less reddened, and in all the acute exanthemata the eruption appears in well-defined places in the characteristic form seen upon the skin.

In general stomatitis catarrhalis we have all the symptoms of an inflammation—swelling, pain, heat, redness. The whole lining of the mouth is hyperæmic; there is more or less puffiness, especially where there is pressure, and here the mucous membrane is somewhat paler. The lips frequently become more tense, and the mucous membrane is covered with small, round prominences due to swelling of the muciparous follicles. When the ducts of the latter become tightly closed the glands dilate, and there are produced cysts, the contents of which are clear, viscid mucus. We also find slight epithelial abrasions, sometimes leading to the production of a deeper process—at all events, important in that they may become the seat of infection.

The tongue is coated, at first dry and white, then yellowish or grayish, and, as secretion increases, whole flakes of this coating are washed off, leaving red spaces partially uncovered. The tongue never looks like the scarlet-fever tongue, since the catarrhal process seems to affect only the superficial layer of epithelium, sparing the fungiform and even the bases of the filiform papillæ. When this process in the mouth is the result of long-continued fevers, the appearance changes; nutrition to all epidermal structures being less active, the tongue and the mouth suffer comparatively more than when the process is purely catarrhal.

In nearly all the inflammations of the mouth the lymphatics become involved, and the intensity of the stomatitis can be measured, as a rule, by the degree of involvement of the glands. Increased temperature is observed (in rare instances as high as 104° F. in the rectum), the prominent symptoms, however, being local. Of these the most important is pain, producing restlessness, fretfulness, and more or less difficulty in nursing. With this, when the child is old enough, there is increased flow of saliva, producing, sometimes, irritation of the skin upon the lower lip or eczema of the face.

Prognosis.—As this is usually an acute process of moderate intensity, the prognosis is good. Indirectly, there may be produced loss in weight, dyspepsia, catarrhal conditions of the intestine, continued enlargement of the glands, possibly tuberculosis, and, therefore, a vulnerability of the mucous membrane, so that the smallest local irritant will be followed by a return of the stomatitis.

Treatment.—In the majority of instances the disease runs its course without any special treatment. The cause must be removed when possible. Next, relief must be given to symptoms; cold water, applied by means of cotton, either wrapped around a stick or the finger of the nurse, or small pieces of ice wrapped in a handkerchief. All food must be given cold; usually this causes least pain; sometimes the opposite will be found necessary. Much comfort will be given by frequent and gentle washing of the mouth with ice-cold sterilized water, to which there has been added boric acid (1–3 per cent.), sodium biborate (2–3 per cent.), zinc sulphate ($\frac{1}{2}$ –1 per cent.), sodium salicylate (1 per cent.), etc. The addition of any of these is not imperative; chlorate of potassium is unnecessary and without value in this form of stomatitis. Silver nitrate ($\frac{1}{2}$ –1 per cent.) is the most reliable of all remedies; if the stomatitis does not disappear in four or five days, the mouth must first be

thoroughly cleaned, and then pencilled with this weak solution once a day. Where there is loss of epithelium the spot should be touched with the mitigated stick, which can be accurately applied by first melting and then dipping a silver probe into it. Cysts should be duly opened, and their walls should be cauterized when necessary.

II. STOMATITIS APHTHOSA.

Aphtha (from *αφθα*, an eruption or ulceration) is a subepithelial vesicle of different color from the mucous membrane upon which it occurs, and is surrounded by an areola which changes in a peculiar way during its existence. It has nothing to do with the muciparous follicles, appearing in places where there are none; it is therefore not follicular.

Etiology.—No uniform local cause has ever been found. Micro-organisms, usually pus-producers, have been observed, but no connection could be discovered between them and the disease. Aphthæ have been produced artificially (caustics, the end of a burning match), but no one has ever succeeded in producing the whole series of symptoms associated with this form of stomatitis. It is said that the disease is most common between the tenth and thirteenth months of life (Bohn), and therefore teething has something to do with the eruption. However this may be, we find stomatitis aphthosa associated with a great number of diseases—pneumonia, ague, gastro-intestinal catarrhs, the acute exanthemata, etc. We must therefore look for the cause in a general, not a local, disturbance, and as the disturbance is the same as herpes, the same etiology will be found to hold good for aphthæ as for herpes. The disease is not contagious, but the same cause may not infrequently produce it in several members of the same family, and usually those are selected whose digestive tracts are either temporarily or permanently weak.

The foot-and-mouth disease in cattle can be definitely accepted as causative, but as this disease is very rare in this country, it can be almost absolutely excluded as an etiological factor. In a recent epidemic near Berlin studied by Siegel, an ovoid bacillus 0.5μ long was found in all cases; only those connected with the animals had local lesions, but were protected in a measure, infection taking place from man to man.

The conclusions arrived at by the author in regard to the etiology of this disease are as follows: It is a disease produced by some form of deleterious material in the circulation, which may have its origin in various processes, bacterial or otherwise. It may, therefore, be of various kinds. This material acts upon a nerve or nerves, or upon a nerve centre or nerve-centres, and produces an herpetic eruption which is the aphthous process.

Symptoms.—On the part of the general system there is a great diversity, depending largely upon the patient affected. We may have, for two or three days preceding the eruption, manifestations pointing to the inception of almost any disease common to children—vomiting, constipation, high fever, pain in the throat or mouth, enlargement of lymphatics, a slight cough, depending upon the localization of the disease, and even nervous symptoms, so that it will be almost impossible to foretell what is coming. On the other hand, some patients are very little affected beyond a slight rise of temperature, fretfulness, and loss of appetite. An examination of the mouth made at this period usually reveals stomatitis catarrhalis, sometimes a whitish spot upon the tonsil. Then, possibly the next day, the characteristic eruption appears with lightning rapidity. This consists of white or yellowish-white subepithelial spots, single or in groups, surrounded by an areola, and developing anywhere within the mouth, not uni-

lateral, and sometimes extending into the pharynx, and possibly also into the larynx. After from twelve to thirty-six hours the epithelial coating is soaked off, and there is left the so-called aphthous ulcer. After a few days more the floor of the ulcer is clean or the exudate is lifted up between regenerating epithelial cells; it is lifted beyond the level of the mucous membrane, and finally disappears. Some aphthæ are absorbed without going through this normal course. They appear in successive crops, and it is not unusual to have the course of the disease extend to from ten to fourteen days. The exudate is made up of fibres, indifferent cells, and various lower forms of life. No cicatrix is left where these spots have been, showing that the submucous tissue has not been affected.

The local symptoms are those of stomatitis catarrhalis; where denudation takes place there is more pain. The most common complication which occurs is stomatitis ulcerosa, and unless this is present the saliva in stomatitis aphthosa is never fetid—a matter of great diagnostic importance. In some instances the aphthæ are so numerous that the mouth looks as if it were covered by a diphtheritic membrane. A day of waiting will clear away any doubts on the subject, as by this time the characteristic denudation will have appeared.

Prognosis.—The prognosis is absolutely good. The disease is self-limited, doing no harm except to interrupt the general thriving of the child. Infection with other poisons has been known to take place, but this, fortunately, is very rare. Relapses are very rare, and the small ulcers, as a rule, heal without difficulty.

Treatment.—This is the same as that used for catarrhal ulcers—viz. the nitrate of silver. Permanganate of potassium may be used locally to great advantage (gr. iiij to f ʒj), but must not be looked upon as a specific. General treatment, as a rule, is not required, and when it is necessary it is purely symptomatic. Laxatives, usually given early, seem to have no influence upon the process; calomel does not abort it, and must be used according to the indications which govern its administration in other conditions. The poison has done its work before we are able to attempt to counteract its bad effects; it is probably eliminated by the time we see the patient, and therefore all causal therapy is futile.

Bednar's Aphthæ are found only in the new-born. They are shallow ulcers covered by a gray or yellowish coating, and found upon the soft palate, the posterior part of the hard palate, the palatine suture, always near the velum palati. They may be mistaken for the ulcers produced by the breaking down of milia or retention-cysts, or for that condition described by Epstein in which there are congenital defects in the mucous membrane filled up with epithelial detritus.

These aphthæ are always produced by violence in cleansing the mouth; this explains their position and their course. They are rarely found in private practice except where the midwife still holds absolute sway. Their course is benign, they require no treatment, and are only dangerous when they become infected. With the modern rubber nipples, when badly shaped, they sometimes develop far forward upon the hard palate; changing the shape of the nipple always results in their cure.

III. STOMATITIS MYCOSA.

This condition, commonly termed Thrush, is a disease produced by a peculiar fungus, first discovered by Berg of Stockholm, and called *oïdium albicans* by Robin. Rees and Grawitz were the first to show that the fungus is not an

oidium, but a *saccharomyces*. All later investigations agree in showing that it is not *oidium*, but all do not agree that it is *saccharomyces albicans*. For the present, however, until the exact position of the fungus is determined, it seems wise to adhere to the last name, *saccharomyces albicans*.

Etiology.—The fungus is the only cause, but it must be deposited upon favorable soil to produce the disease. The *saccharomyces albicans* may be found upon every mucous membrane in the body, the alimentary, the respiratory, and genito-urinary: it has been found in the parenchyma of organs, as the brain and lungs, and in blood-vessels. It is usually carried to children by the nipple or by the nursing-bottle. The fact that weak and unhealthy children are most predisposed to thrush has been emphasized entirely too much: perfectly healthy children have thrush. It has also been stated that flat epithelium is necessary for the development of thrush; this, however, can no longer be maintained, as we see the fungus on a great many surfaces lined by cylindrical epithelium. It is admitted on all hands, however, that catarrhal stomatitis exists either before or with the appearance of thrush. It is more than probable that this is the predisposing cause, and that it works mechanically—viz. by a dislocation of the swollen cells, preventing perfect protection to the mucous membrane, and allowing the spores of the fungus to find a place for development. Anything producing this mechanical injury to the membrane of the mouth, such as badly-formed or hard nipples, will act in the same way. The younger the child or the weaker, the more successful will be the implantation of the *saccharomyces*, because the function of motion of the tongue and jaw will be least developed. The disease is therefore found especially in infants reduced by illness, and in older children in connection with diseases that are followed by great loss of strength, such as long-continued fevers, wasting diseases, or those in which motion is very much impaired.

The fungus is found in two forms, depending largely upon the culture-material—the yeast form and the globulo-filamentous form (frequently called mycelium). There is no ascospore; therefore, according to Roux and Linoissier, the fungus is not a *saccharomyces*. The chlamydospore has, however, not been satisfactorily worked out. Propagation goes on in three ways—by filaments produced from conidia, by isolated conidia, and by spores.

Pathology.—The first lodgement comes between the epithelial cells of the mouth, and from this the growth works its way toward the free surface and toward the mucous membrane proper. In the direction of the free surface the growth is not so luxuriant, but in both directions it is principally in the mycelium form. In mucous membranes lined by flat or squamous epithelium the growth of the *saccharomyces* is facilitated by the relation of the cells to each other; in membranes lined by cylindrical epithelium growth takes place, but not so readily, because there is but one layer of cells. After the first development growth goes on very rapidly: after having found a nidus, the cells are pushed aside, surrounded by mycelium, the whole forming the characteristic thrush-spot. Pus is rarely produced; when this does occur the affection is of a complex nature. The growth begins in small spots, sometimes one, sometimes more; from these infection spreads, and at times the whole mucous membrane is covered with a rich growth of the *saccharomyces*.

Symptoms.—Preceded or accompanied by stomatitis catarrhalis, the local symptoms vary with the intensity of this process. Frequently no symptoms are present, and the existence of the small spots is the first indication of the presence of thrush. These vary in size, seem a part of the mucous membrane, are usually of a grayish-white, creamy color, and may or may not be elevated above the surface of the mucous membrane. They appear first upon

the tongue and cheeks, then frequently upon the lips and soft palate, and may be found upon the tonsils, the pharynx, or the œsophagus. With only moderate care of the mouth they seem to last indefinitely; without care they spread rapidly, and instead of the spots we may see membranes, in the case of the œsophagus whole casts being formed, which fill its lumen and often prevent swallowing. In hospital practice thrush has proved a formidable disease; in private practice it amounts to nothing more than a local disturbance, unless neglected. In the latter class of patients there is always associated some gastro-intestinal disturbance, which may prove serious if not fatal. In debilitated subjects—and thrush, from the mechanical reasons pointed out before, is more common in such—these gastro-intestinal troubles may be the affection which terminates the child's life. When the membrane drops off there is left a slight abrasion which may become the focus of infection by any other morbid agent.

But it must not be inferred that thrush occurs only in debilitated or sick children. It may occur in children that seem perfectly healthy, although careful investigation will always reveal some lesion in the mouth which has preceded the thrush. Again, not every child with stomatitis mycosa has gastro-intestinal symptoms: the food carrying *saccharomyces* frequently carries other lower forms of life capable of producing diarrhoea, but in properly-treated cases these symptoms are wanting, and when taken early enough thrush is local, and local only.

The thrush-spots develop within the epithelium, and examination by reflected light will show this; the spot is often surrounded by a narrow ring of injected blood-vessels. Removal from the mucous membrane requires considerable violence. The next step in development is a pushing up beyond the level of the mucous membrane, and after this more extensive infection of the mouth may be expected unless counteracted by treatment. At times the whole mass may drop off and leave an ulcer, sometimes very intractable, or the many spots may coalesce to form a membrane. The differential diagnosis is not difficult if all the above be taken into consideration, and a positive diagnosis can be made under all circumstances with the microscope.

Treatment.—Prophylaxis is very important. In young children all abrasions and all slight forms of stomatitis ought to be looked after. Everything coming in contact with the mouth of the infant should be kept aseptic—the nipples, the feeding-bottle, the food.

The treatment is simple enough if properly carried out. A solution of sodium bicarbonate (1 drachm to a tumbler of water) is to be applied with a brush between the times of nursing or feeding and immediately after feeding or nursing. Ulcers should be treated as has been described under Stomatitis Catarrhalis. Œsophageal thrush, when the diagnosis is possible, should be treated by the introduction of a soft-rubber tube (catheter) into the stomach. The intestinal troubles are best treated by small doses of calomel or corrosive sublimate, combined with careful diet.

IV. STOMATITIS ULCEROSA.

This is a peculiar process, characterized by destruction of tissue, beginning on the gums around the teeth, never extending beyond the mouth, infecting healthy parts of the mouth, and never occurring where there are no teeth.

Etiology.—A clinical picture resembling the disease is produced by the internal administration of certain remedies—mercury, copper, and iodine. Mercurial stomatitis is almost identical with stomatitis ulcerosa, and in these

cases we find that a local irritation caused by bad teeth or uncleanness of the mouth is a decided factor in the production of this affection. But, in addition, the remedies are excreted by the mouth, and in this fact there is to be found a possible clue as to the etiology of stomatitis ulcerosa. Whether, in addition, there are lower forms of life or chemical substances, or both, which cause this peculiar form of inflammation, it is for the present impossible to decide.

The disease usually develops in connection with bad hygienic surroundings, or following certain diseases, especially measles and scarlatina, and frequently malaria, pertussis, typhoid fever, or pneumonia. It is said to be endemic in the wards of certain hospitals or in certain barracks; and damp, poorly-ventilated houses, with or without insufficient nourishment, certainly favor its development. The disease is usually held to be non-contagious, but experiments with inoculation have proven to me that, with proper precautions, the disease can be propagated. It is not infrequent with soldiers, especially when confined in barracks, and the likelihood of a scorbutic affection being the predisposing factor cannot be disposed of at present. It is rarely observed before the age of five years, most frequently between the ages of five and ten, but it does occur at any time of life, provided teeth be present.

Pathology.—The process is one of necrobiosis. There is cellular death, but at the same time there results softening of the tissues, and not death *en masse*. The peculiarity of this form of necrosis is that it does not respect any form of tissue, but may extend to the periosteum, finally producing necrosis of bone. It is not unusual to find sequestra of large size ready to be removed. The process may, at the same time, produce caries of the bone, although this is certainly exceptional. The disease always begins at the free border of the gums, from which it extends in all directions, frequently infecting healthy mucous membrane, but never extending beyond that of the mouth.

Symptoms.—We first find swelling of the mucous membrane only at the lower part of each tooth (most commonly the lower incisors), and this gradually increases until the curved outline of the gum is converted into a more or less straight line. This swelling may become so great as to produce eversion of the part affected; at the same time there is great injection, almost lividity, accompanied by more or less bleeding upon the slightest provocation. The anterior aspect of the gum is first affected, but in severe cases the posterior portion also takes part in the process. Soon the gums can be detached from the teeth, and there is exposed a cavity or sac filled with a muco-purulent secretion. These characteristic local symptoms are further distinguished by the appearance upon the swollen gum of a yellowish seam, which may become a broad band. This represents the ulceration, and is due to cellular necrosis. With this there is a constant flow of fetid saliva from the mouth, but the odor comes from the diseased gums, except in very bad cases, when it may in part occur from diseased bone. In older children subjective symptoms are slight; in younger ones the principal evidence is pain, fretfulness, change in disposition, crying, and wakefulness.

The outpouring of large quantities of saliva commonly produces eczema of the lips, which may persist long after the cause has been removed. The lymphatic glands are always involved; they are soft, and remain enlarged frequently for a long time; as a rule, they do not suppurate, although this may occur some time after the disease in the mouth has run its course.

At this stage the disease is very amenable to treatment; if left to itself, it goes on indefinitely and develops. The yellowish seam increases, and when removed there is exposed an ulcerated surface. There is greater formation of

pus; the gums become more detached from the teeth, which are loosened. Ulcers may now form upon other parts of the mouth, the lips, the cheeks, the tongue. In very bad cases the whole of the mucous membrane covering the body of the lower gum has ulcerated away, and we look in upon a cavity filled with offensive pus, bleeding, and possibly showing a piece of denuded bone at the bottom. In these cases salivation has reached its maximum development, and the whole room may become tainted with a peculiar foul odor. Sometimes ulcers form upon the mucous membrane joining the lower lip to the gum; wherever they may be, however, it is always the characteristic sequence: first, necrobiosis, the seam surrounded by injected tissue, then ulceration below.

Nature rarely cures these cases without assistance: when cure takes place the symptoms disappear slowly, but in every case the disappearance of the fetid saliva is the first symptom of improvement. Sometimes the disease becomes chronic; it then runs an exceedingly mild course when deep tissues are not involved. It always takes some time for this to take place, so that if a patient has had stomatitis ulcerosa for several months without involvement of deep structures, it is more than probable that we are dealing with the chronic form. This is characterized by its resistance to ordinary methods of treatment and by the frequency of relapses.

Prognosis depends upon three factors: the disease upon which stomatitis ulcerosa is engrafted, the stage of the disease, and the treatment. The worst form is found in scorbutus. Where bone-changes are present the disease assumes the aspect of a disease of bone, but the prognosis is not bad when the condition is recognized. The important fact that stomatitis gangrenosa sometimes develops must never be forgotten: every case of stomatitis ulcerosa, therefore, requires most careful watching.

Treatment is prophylactic and curative. Improve the hygienic conditions of the patient and prevent extension of the disease to others. Chlorate of potassium can be looked upon as almost a specific in this affection. It is to be administered, with all precautions, in a 3 per cent. solution, of which $\frac{1}{2}$ to 1 teaspoonful is given every two hours. At first its administration is accompanied by pain, sometimes very intense, but this no longer occurs in from thirty-six to forty-eight hours after treatment has begun. It takes about twenty-four hours for the remedy to produce any appreciable effect, and this is evidenced by a diminution of salivation. Soon this hypersecretion disappears entirely, and with it the fetid odor from the mouth; in the course of a week, usually, all symptoms will have disappeared. If ulceration has not disappeared at this time, careful search must be made for the cause. Carious teeth must either be removed, filled, or otherwise treated by antiseptics; if this does not remove the ulceration, recourse must be had to cauterization, either by nitrate of silver or the galvano-cautery. Dead bone must always be removed. Where the cause of a continuance of the process cannot be found, frequent applications of permanganate of potassium yield good results. As a last resort, the teeth around which the ulcerative process is best developed must be extracted and the cavity frequently washed, when the process will soon be found to come to an end. As potassium chlorate is a remedy almost specific in its properties, any other medicaments will hardly ever become necessary.

In chronic cases potassium chlorate does not act so universally; here, however, its use is also indicated, combined with local treatment in the form of applications of silver nitrate three times a week.

V. STOMATITIS GANGRÆNOSA.

This disease, termed also *cancrum oris*, gangrene of the mouth, or *noma*, is comparatively rare, most common in hospital practice, and in private practice depends for its frequency principally upon the surroundings. It is a gangrenous process, beginning upon the gums or inner surface of the cheek, spreading with great rapidity, and destroying every kind of tissue upon which it develops.

Etiology.—There can be no doubt that the disease may become infectious in its nature; several cases occurring in the same family or a number breaking out in one ward of a hospital can be offered in evidence. On the other hand, a great many cases are observed in which it seems to be impossible to take into consideration anything like contagiousness; a case occurring in a patient miles out in the country where no other case of *noma* has ever been observed in that neighborhood.

Noma occurs only in children sick with other diseases, never in healthy children. Furthermore, it follows in the wake of such diseases as produce great debility and least cellular resistance. The diseases most commonly followed by *noma* are—the acute exanthemata (especially measles and typhus), whooping-cough, syphilis, scorbutus, chronic intestinal catarrhs, and malaria. The excessive use of mercurials has been frequently considered the cause of this disease; no doubt such consequences have followed the heroic doses of former days, but are certainly exceptional now. *Stomatitis ulcerosa* is frequently a forerunner of *noma*, being the result of identical predisposing causes, but in all probability the resemblance of the two processes ceases there.

A great many lower forms of life have been found, but the testimony as to their causative relation is, as yet, inconclusive. Short rods, as in pulmonary gangrene, and streptococci (Cornil and Babes), streptococci resembling those found by Koch in progressive tissue-necrosis of white mice (Ranke), and bacilli in thread-like growth (Lingard), have been looked upon as the immediate cause; but the predisposing cause, after all, is the most important, and the probability is that sooner or later any number of different kinds of organisms, both pathogenic and otherwise, are developed in every case of *noma*.

Pathology.—We find all the evidences of a phlegmonous gangrene. Surrounding destroyed tissue there is an infiltrated zone. The latter is a true necrobiotic process, all evidences of cellular tissue being destroyed, only a homogeneous substance in which are found micrococci being left. Around this is found increased connective tissue, the connective-tissue corpuscles in active cell-division, while the blood-vessels are closed by thrombi and lower forms of life. Outside of this we find healthy tissue. In every case of *noma* these four zones can be distinguished.

Symptoms.—The first and most characteristic symptom noticed is the odor of gangrene. Upon examination an ulcer will be found upon the gums or the inner surface of the cheek; this spreads very rapidly. Very soon the whole cheek begins to swell; it becomes œdematous, the skin is waxy, and within twenty-four hours the whole side of the face may become involved. Sometimes the swelling is painful, but frequently children will not complain of any local symptoms. The ulcer in the mouth has now become deeper, and is evidently eating its way through the cheek, producing symptoms almost pathognomonic as it comes nearer the skin. The latter becomes discolored, red, blue, purple, black. Sometimes a bulla filled with ichorous fluid is formed upon the cheek; then the epithelial covering breaks down, and with it the gan-

grenous process goes on from without inward. Where no bulla is formed the gangrene goes on from the mucous membrane to the skin. Perforation of the

FIG. 1.



Cancerum oris in a child five years old.

cheek takes place under all circumstances and in a very short time—from twenty-four hours to three or four days. As a rule, the process continues, involving the whole of the cheek, the neck, the eyelids, destroying the eye, but rarely becoming bilateral. The bones are denuded, the teeth become loose, the tongue, hard and soft palate, even the tonsils, may become infected, and there is left a discolored, fetid, soft mass. The whole terminates in producing probably the most repulsive appearance the physician has opportunity to see. The odor is frightful, filling the whole house; the flow of saliva is very much increased, and death usually results from the depressed general condition. Spontaneous recovery is rare: a line of demarcation then forms around the gangrenous spot, the surface is covered by granulations, and finally cicatrization follows, leaving most horrible scars. Relapses sometimes occur, but they are rare. The whole duration of the disease is from one to three weeks, sometimes longer, depending upon the vitality of the patient. The general symptoms are usually those of the disease upon which noma is engrafted. Sometimes children with noma are found playing in bed, picking out loose teeth, and apparently little concerned about the intense fatal process going on upon their cheek. This is, however, not the rule, and when it does occur it is followed in a short time by general symptoms showing the severity of the local process. The temperature is sometimes very high, becoming hectic in type, but not infrequently it becomes subnormal before death. The pulse is small, easily compressed, weak, and rapid. The appetite is diminished, and diarrhoea is the rule, most intractable in its nature and probably due to infection from the process in the mouth. Catarrhal pneumonia, due to inhalation of septic material, is common, and diphtheria

has been observed in several cases. Exhaustion comes on, and then the child becomes apathetic, refuses food, and dies in collapse. Hæmorrhages are rare, because the blood-vessels are filled with thrombi.

Prognosis.—This is very bad, the mortality ranging from 70 to 90 per cent. of all cases affected. Complications make the prognosis absolutely fatal.

Treatment.—Of the general treatment, always of great importance, little new can be said, as the physician has already done all in his power to avert a gangrenous process by keeping up the strength of the patient. When noma sets in stimulants should be used methodically and systematically; food should be given in as condensed a form as possible. If feasible, rectal alimentation may be tried, but this, as a rule, is not very satisfactory for children.

The local treatment is of prime importance, and, as the mortality is so great, even the most heroic treatment can be adopted with complacency. The principle of local treatment is to destroy the infiltrated zone and the healthy tissue surrounding it for some distance, so as to make an artificial line of demarcation. Nitrate of silver in stick, dilute muriatic or other acids, chloride of zinc, and many other remedies have been recommended for this purpose. To the author it seems that the best and most active method of destroying this tissue is to be found in the use of the thermo-cautery of Paquelin or the galvano-cautery; and lately several cases have been reported in which success has followed these applications, although it is far too early to draw positive conclusions. As soon as the gangrenous nature of the disease has been established the operation must be performed. A loss of time, even of hours, means considerable loss of tissue. Again, delay may make the operation one of great magnitude, in that blood-vessels may have to be tied which before the extension of the process could be safely cut with the galvano-caustic knife. Under anæsthesia, when possible, necrotic tissue should be removed, and then everything that seems gangrenous should be destroyed. After this a certain amount of healthy tissue should be cauterized. If gangrenous spots appear the next day, the operation should be repeated, and so on; applications can be made daily. The wound is to be treated according to surgical rules, and plastic operations should be put off as long as possible, because, in the first place, they do not offer much chance of success when done early, and, in the second place, noma sometimes recurs as the result of these operations.

In conclusion, it must be stated that, whatever has been done and will be done, the results must be bad, because the process is one developed in a patient very much reduced, in whom the least complication is likely to prove fatal.

VI. STOMATITIS CROUPOSA; STOMATITIS DIPHThERITICA.

Croupous stomatitis may be produced by a variety of causes, both chemical and bacterial. Primary croupous stomatitis is certainly a very rare affection, although it may occur. As a rule, the croupous membrane develops contemporaneously with a membrane upon the tonsils. In very severe cases the membrane has been found upon the cheeks, the tongue, and even upon the lips. The lymphatic glands are not much involved, and as the mouth-process is commonly only part of another of more importance, little more will be said in this connection. The important thing to establish is the absence of the Loeffler-Klebs bacillus; this will make the diagnosis absolute. At the present time the whole subject is being investigated, but enough has already been done to show that all false membranes are not diphtheritic.

Diphtheritic stomatitis does occur as a primary affection, although it is

not very common. When primary in the mouth, the membrane usually develops upon the lips, and may extend thence to any part of the mouth. As a rule, the tonsil is the primary seat, and thence the membrane spreads to the soft palate, the tongue, the cheeks, the lips, and the gums. There is but one positive method of making the diagnosis of diphtheria, and that is by proving the presence of the Loeffler-Klebs bacillus by cultures, and then making inoculative experiments upon lower animals. In primary diphtheritic stomatitis this would become imperative; in the secondary form there are, fortunately, still left for the clinician combinations of certain symptoms that make it possible to diagnose the disease without consulting the bacteriologist.

Salivation usually occurs, and the odor from the mouth is fetid. Sometimes diphtheria of the mouth, when primary, runs its course most insidiously, and is overlooked or not recognized until further complications develop. The membrane lasts from three to six days, sometimes longer, and then either drops off or ulcerates away; in either instance there is left a denuded place. Hæmorrhages are common, either slight or otherwise; when not due to mechanical irritation they are matters of anxiety. In some instances hæmorrhage has been so great as to cause death; in others only a slight loss of blood seems sufficient to produce a fatal termination. The prognosis depends largely upon the form, whether primary or secondary; it is very much worse in the latter than in the former, but even in the primary form may become very grave by extension. The author has seen two cases in which a primary diphtheritic stomatitis has become a laryngeal one.

Treatment is that of diphtheria. When possible, the membrane must be removed if this proceeding be not accompanied by violence, so that infection of healthy membrane be produced. Constitutional treatment is of the utmost importance, in order to counteract the tox-albumins produced by the bacillus. For this purpose corrosive sublimate, administered internally in full doses frequently repeated, seems to be the favorite. In the septic cases much good can be done by frequent local applications without violence.

VII. STOMATITIS SYPHILITICA.

Syphilis produces stomatitis only in an indirect manner, either by causing a specific deposit, which, in its turn, produces the disease, or by rendering the mouth in such patients more sensitive to agents which produce stomatitis.

The three stages of syphilis are developed in the mouth. Primary lesions are very rare, but infection does take place from syphilitic wet-nurses, and when this occurs the lesion in the mouth of the child does not differ from the same lesion in the adult. The secondary manifestations are most common, and any part of the mucous membrane may be their seat. Upon the lips we find the following forms: syphilitic fissures, papules, plaques, and erosions. The fissures (rhagades) are most common, and are generally found at the corners of the mouth or upon the upper and lower lips. They are syphilitic infiltrations which have been split near their middle, so that at the corner of the mouth one part of the infiltration lies nearer the upper lip, the other nearer the lower, and the split seems a continuation of the commissure. Upon the lip rhagades usually end in the mucous membrane. Sometimes these fissures are present in such great numbers that they disfigure the mouth, and by the pain which they produce cause great annoyance to the patient. When they heal they leave cicatrices which, in their turn, may permanently disfigure the mouth. The characteristics of these fissures are the infiltration, the split, and the lack

of tendency to spontaneous healing. Papules are most common at the commissure and the free border of the lips; they may also be split, and then resemble the former variety. As a rule they look like condylomata lata in similar positions; they are elevated, their surface is moist, the centre has a tendency to break down, and unless they involve the mucous membrane they do not cause pain. The remaining forms may be found upon any part of the mucous membrane; they cover more space, are not characterized by the same amount of infiltration, but usually produce more pain and more salivation.

Upon the tongue we most commonly find plaques muqueuses and syphilitic ulcers. Their localization depends largely upon irritation, either from a sharp tooth or other cause. The healed ulcers leave cicatrices, but the characteristic appearance of the tongue, as it is found in the adult after syphilis has run its course, is exceedingly rare in children. In the early stages of syphilis we find a decided enlargement of the circumvallate papillæ, and a loss of the filiform papillæ, so that the tongue looks "shaven." The so-called geographical tongue (wandering rash, ringworm, lichenoid condition) has nothing in common with syphilis and bears no relation to it.

Treatment.—As in all forms of syphilis, so with stomatitis syphilitica—general treatment is of most importance. When deformity or danger to life is threatened, that method must be used which produces the quickest effects. The manifestations in the mouth, as a rule, yield rapidly to constitutional treatment, but local prophylaxis and treatment must not be lost sight of, as being accessory and highly important. Cleanliness is absolutely necessary to prevent salivation as well as to aid in recovery. All sources of irritation must be removed and the teeth must be kept in good condition. Frequent applications of silver nitrate are best for ulcers, erosions, or losses of substance. Corrosive sublimate is preferable when there is considerable infiltration, either in weak solution as a mouth-wash, or in stronger solution applied with a brush, in which case it is apt to produce pain. The weak solutions should be applied two to four times daily; the strong ones (as high as 12 per cent.) are caustic and should be used with great caution. When children are old enough an application of emplastrum hydrargyri with lanolin (1 part of lanolin to 2 parts of the emplastrum) upon chamois gives better results than either of the former remedies in rhagades at the corners of the mouth. In cases of stomatitis mercurialis, potassium chlorate or any remedy containing tannic acid, such as tannin itself or tincture of rhatany or catechu, is very serviceable.

II. DENTITION.

NEARLY all diseases of childhood have been ascribed to teething; even at the present time authors will be found who do not hesitate to work out the most improbable relations of teething to disease. But, be this as it may, there is no one who does not admit that some children may have teeth without any great amount of disturbance, or, indeed, that teething may go on without producing any symptoms at all. This latter form of teething would be called normal; the abnormal form has been called *dentitio difficilis*. It is proper to state that medical authorities are much divided as to the importance of teething as an etiological factor of disease, and that they can be divided into three classes: those claiming that almost any disease can be produced by teething,

those claiming that no disease is produced by teething, and, lastly, those who state that some few diseases may follow the eruption of teeth. The first class states that normal teething occurs in only 20 per cent. of all children. Although teething in healthy and teething in unhealthy children is a better division from a clinical standpoint, we will, for the present, follow the division as given above.

The greater part of teething is accomplished before the child is born. At about the seventh week of foetal life the epithelium within the mouth is thickened, forms a ridge, and at the same time dips into the embryonic tissue about to form the jaw. This epithelial process is called the enamel-germ; it grows so as to surround a flask-shaped cavity, which it lines; partitions develop into this, forming ten cavities for each jaw. A papilla is now developed, which, pushing up toward the embryonic tooth, forms a complete mould for the enamel-germ to rest upon, and this is called the dentine-germ. We now have the beginnings of the ten temporary teeth in the form of the partitions, the enamel- and dentine-germ, and the papillæ. The connective tissue around these primitive teeth has at the same time been forming into the dental sac, an investing membrane for each tooth. In the partitions, as well as in the rest of the jaw, bony tissue is being formed; the teeth become farther separated from each other, and by this deposit of bone the alveolus is formed, lined by the dental sac coherent with the gum along the border of the jaw. This process of development has taken the whole period of foetal life, so that the child comes into the world with all its temporary teeth fully formed within the jaw. The permanent teeth are formed, in so far that the enamel-germ is developed from the enamel-germ of the temporary tooth as a small sac, from which subsequently the development goes on, as already described for the temporary teeth. The topographical relations of the teeth at birth are as follows: above, the tooth-sac, the submucous connective tissue, and the mucous membrane itself; on either side, the tooth-sac and bony tissue. There is no bony tissue to impede the tooth on its way to the oral cavity; all that it needs to overcome is the submucous coat, the mucous membrane, and the dental sac, which is very thin. Not enough stress can be laid upon the fact that the opening of the alveolus is wider than necessary to allow the tooth to pass through.

Calcification of the fangs begins, and as the tooth becomes elongated by means of this, it is slowly forced in the direction of least resistance, the mouth. Pressure is directed toward the mouth; the papilla cannot be pressed upon, for the simple reason that where, during growth, blood-vessels come in contact with bony substances, absorption of the latter is produced, the blood-vessels not being affected. It is possible that, as Kassowitz has pointed out, the growth of the blood-vessels causes the alveolus to be moved constantly, and that this growth acts as another cause for the coming through of the teeth. Calcification of the fangs usually begins in the lower incisor teeth at birth, beginning in those teeth first which are first to make their appearance in the mouth.

The order of teething can be described as occurring in three ways. Unfortunately, there is as yet no unanimity among authors as to the most common method. The first is the appearance of the teeth in pairs, principally in relation to the incisors. The second is the appearance of the first *two* incisors, then all the other incisors, and then the molars. The third, which we believe to be the most common order, is the appearance of the first two lower incisors, then the four upper incisors, then the first molars, and with them the remaining two lower incisors, as follows:

I. Two lower central incisors	5-7 months.
II. Four upper incisors	8-10 "
III. Four first molars and two lower lateral incisors	12-14 "
IV. Four canines	18-20 "
V. Four second molars	28-34 "

It will be borne in mind that this table represents average times, and that the time for eruption depends upon a great many different causes. The nationality, heredity, climate, and general development of the child may either retard or accelerate the appearance of teeth. Certain diseases, especially rickets, have a well-marked retarding influence, but because a child is late in teething it must by no means be taken as positive evidence that he has rickets.

The time of eruption depends, first, upon the distance the tooth has to travel from the dental sac to the mouth; secondly, the amount of calcification in the fangs; and, lastly, the condition of the rudimentary organs. Increased calcareous deposit would compensate for length of distance, and possibly for deficiencies in the rudimentary organs; but frequently no compensation can take place, and the teeth are left permanently deformed as well as late in appearing.

Premature teeth may occur from several causes: some change in the embryonic structure may result in the production of teeth without fangs, which are attached only by mucous membrane; or the deposit of calcareous material may be too early or too great; or, finally, more than twenty primitive teeth may have been formed, one or more of which project into the cavity of the mouth. Premature ossification of the bones of the skull is said to be accompanied by premature teeth, and in this case Jacobi claims that the upper incisors then appear first. The latter view, however, still requires verification. Premature teeth must not be interfered with unless there is a special indication for their removal, because it may be possible that no second tooth shall appear until the permanent one comes through; and, furthermore, their removal is not unattended by danger (hæmorrhage). The most urgent indication for removal is to be found in their being in the way of nursing; they may produce fissure of the nipples or may make nursing so painful to the mother that serious consequences follow.

The teeth are retarded by the constitutional diseases, rickets and syphilis—these forms of general disturbance of nutrition resulting in cachexia and in long-continued fevers or chronic diarrhœa. Acute febrile disturbances, such as the exanthemata, may not have any effect upon the temporary teeth, and yet show distinct tracings upon the permanent teeth; or the group coming through at the time of fever may not be delayed at all, and yet the next one will be delayed some time.

A food-supply defective in calcareous material has been frequently accused of delaying teething. This is, theoretically, correct; but, as a matter of fact, when the salt material of the food is diminished to such an extent as not to be able to supply the small amount demanded for teething, life can no longer be sustained by such food. Our own experience has been that none of the proximate principles of which teeth are composed, when administered internally, have any effect upon the appearance of the teeth. There is but one remedy which seems to hasten teething, and that one affects rachitic children principally, though not exclusively; we refer to the internal administration of phosphorus.

The permanent teeth appear in about the following order and times:

First Molars.	Incisors.	Bicuspid.	Canines.	Second Molars.	Third Molars.
6 years.	7-8 years.	9-10 years.	12-14 years.	12-15 years.	17-25 years.

In regard to the symptoms produced by teething, it can be definitely stated that in a healthy child teething goes on without producing symptoms of any sort. In children reduced by malnutrition, affected by hereditary syphilis or rickets, and in those extremely nervous either as a result of hereditary or other causes, there are symptoms which can be divided into two groups: first, local; secondly, remote. The local symptoms are pain, heat, irritation, not infrequently stomatitis catarrhalis. All these may occur in healthy children, but are manifestly of little importance, as they produce little if any general reaction, and are certainly very rare. At times children may become a little fretful or cross, and in the evening have a slight rise of temperature. As a rule, however, the teeth which have long been expected by the anxious watchers make their appearance without premonitory signs, so that the wise physician will hesitate before he prophesies when a tooth is to appear. Salivation cannot be looked upon as a symptom of teething, as it usually occurs from two to three months before the first incisors appear, and is physiological. The salivation occurring during teething is due to stomatitis. The pain can only be very slight, and can be judged by analogy with that produced during the appearance of the second teeth. In an unhealthy or over-sensitive child this, however, may be sufficient to produce restlessness or peevishness. That the pain cannot be very great must be accepted also from anatomical facts: the nerve-filaments covering the tooth have either been absorbed or rendered insensitive by continuous pressure upon them. The papilla cannot be taken into consideration at all, as it has been shown that the teeth could not in any way press upon it.

The symptoms in remote parts have to be analyzed carefully, and much cool judgment may be required to find their cause. The tendency at the present time is to accept fewer and fewer symptoms as due to teething; but for convenience we have grouped them under the following headings: symptoms on the part of the nervous system, the digestive apparatus, the skin, the respiratory apparatus, the genito-urinary system, and the organs of special sense.

The principal symptom on the part of the nervous system, still adhered to by many, is convulsions. It is claimed that they are of a reflex kind, the tooth being the irritant producing an abnormal afferent impulse to the medulla. Theoretically, this can be taken into consideration, but in practice convulsions are not produced by teething, least of all as the result of a reflex mechanism. Tonic contractions of muscles of a local nature may easily be produced by an increased afferent impulse, but the most painful lesions involving the fifth pair of nerves in the reflex arc are not followed by generalized muscular contractions. In the alimentary canal we find the bowels participating in the general hypersensibility of the child. There is no evidence to show that bowel lesions are produced by teething, either as the result of swallowing an imaginary excess of saliva or otherwise. The most pernicious doctrine that exists is the one that intestinal disease is due to teeth. An over-fed or badly-fed child—and at the time of the eruption of the canines it is most liable to be both—if suffering, generally has an irritable intestine; and very likely substances which should not enter the circulation may pass into it from the intestine, and the result will be stools changed as to quantity and quality. This, in the latter instance, is a curative act, and disappears as soon as the diet is corrected. There is nothing characteristic about this form of diarrhœa; it rarely becomes pathological, and may be helped along by the administration of a laxative. Any diarrhœa, however, occurring at any time during infancy should be

watched, whether the child is supposed to be teething or not, and, the cause being removed, the bowels should be "checked." It is important to disregard teething entirely in long-continued diarrhœa, and to look to the food or other known agencies for the cause.

On the part of other organs the symptoms which occur must be looked upon as concomitant with teething and not caused by it.

Some have claimed that teething does not, *per se*, make children sick, but that it predisposes them to illness. Predisposition to disease undoubtedly exists, both temporary and permanent, but it is a difficult thing to establish, and, from what we know at present, such a theory must be denied absolutely as far as teething is concerned.

There is no treatment for teething, as it requires none. The healthy child has no symptoms to manifest any diseased condition, because there is no disease. The unimportant symptoms that may occur are to be treated purely symptomatically. The restlessness, where necessary, will be relieved by bromides. The various forms of stomatitis are to be treated by the appropriate remedies referred to in another place. Bowel troubles require rigid diet, always a proper precautionary measure in all forms of intestinal disturbances. Beyond this nothing is required.

Gum-lancing or gum-scarifying is looked upon by many as the specific method of treatment for teething ailments. The indication for the operation is to relieve pressure. The tooth has been supposed to press in any or all directions, and by means of this pressure to produce the baneful results referred to. Some authors claim that the pressure is exerted upon the mucous membrane; others, upon the dental sac; others, upon the alveolus; and finally others, upon the "sensitive" papilla. Accordingly, each one has a peculiar method to recommend for the operation. From a practical standpoint any of these methods can do good in only one of two ways—either as a method of blood-letting or as a suggestive remedy; but either indication can be met by simpler means. From a theoretical standpoint everything is against any such method of operation. It has already been shown that the papilla cannot be pressed upon, and that the opposite condition exists: the papilla is forcing the tooth. All this in the growing tooth is done so gradually, however, that very little pressure is exerted in any direction. The mucous membrane cannot be accused of suffering, for, as we have seen, movement of the teeth toward the oral cavity practically begins at birth. Given any mucous membrane which has been pressed upon by a rigid substance for from five to seven months, and atrophy will undoubtedly follow—atrophy of the membrane and all its component parts, including the nerves. For the same reason pressure upon the dental sac would be impossible. Pressure upon the bony walls is out of the question, because there is ample room in all directions for the tooth, the opening of the alveolus being especially large, so that the crown of the tooth can pass without difficulty.

It cannot be denied that indiscriminate gum-lancing does harm. Hæmorrhage is its greatest danger: we have collected twelve fatal cases, and it is not overstating the matter when we say that many more have occurred that have not been recorded. Behrend, Churchill, Barthez and Rilliet, and Finlayson refer to the danger to children arising from anæmia as a result of this operation—a danger that cannot be expressed statistically. Under normal circumstances the pushing through of a tooth does not leave a wound of any sort: there are no lymphatics, no blood-vessels opened, these having been closed by the process referred to before. Every time a gum is lanced an open wound is produced—fortunately, one which, under ordinary circumstances, heals

quite rapidly. But with the mouth as a playground for many pathogenic microbes the danger of infection must not be under-estimated.

In conclusion, I wish to emphasize the following points: I. Gum-lancing is useless, *a*, as far as giving relief to symptoms; *b*, as far as facilitating or hastening teething. II. It is useful only as bloodletting or as a suggestion, and ought not to be used as such. III. It is harmful, *a*, in producing local trouble; *b*, in producing great disturbance on account of hæmorrhage; *c*, in having established a method which is too general for specific good and too specific for universal use. IV. It is to be used only as a surgical procedure to give relief for surgical accidents.¹

¹ The author certainly presents in a very forcible manner one side of the disputed question of the advisability of gum-lancing. That too many aberrations from health are laid to the score of teething, and that lancing is often performed heedlessly, unnecessarily, and even injuriously, cannot be questioned, yet there are many well-informed physicians and clinicians who use the lance in appropriate cases, because experience—the crucial test—has demonstrated its utility. In this class the Editor must be included.—L. S.

DISEASES OF THE PHARYNX AND NASO-PHARYNX.

BY W. E. CASSELBERRY, M. D.,
CHICAGO.

I. ACUTE PHARYNGITIS AND NASO-PHARYNGITIS.

THE posterior wall, the vault, and the lateral angles of the pharynx, the pillars of the fauces, the velum palati, and the tonsils may be, each alone or all together, the seat of an acute inflammation of the mucous membrane, which for convenience is commonly designated simply as "pharyngitis."

Predominant inflammatory diseases of the tonsils, however, are considered apart under appropriate titles, although tonsillitis of a superficial type is often a detail only of diffuse simple pharyngitis, and may then be included in the latter term. The forms of symptomatic pharyngitis which are incidental to the exanthemata are excluded from consideration at this point.

Etiology.—The predisposing conditions are chronic hypertrophy of the faucial and naso-pharyngeal tonsils, acute or chronic rhinitis, previously existing chronic pharyngitis, and digestive disturbance. Climatic inequalities, with exposure to chilling influences, furnish adequate exciting causes.

Pathology and Symptoms.—Hyperæmia may be so pronounced and so diffuse as to lend a bright reddish hue to the entire oro-pharynx, or, on the other hand, only limited spots of congestion may be noticeable. Often the pillars of the fauces alone are implicated.

The posterior surface of the velum palati is a frequent point of attack, and, indeed, the disease not infrequently embraces the rest of the naso-pharynx, and occasions an amount of pain and discomfort located high up which is far in excess of that which can be explained by inspection of the fauces only. More explicitly speaking, naso-pharyngitis may be conjoined with pharyngitis.

After the first twenty-four hours thickening and relaxation of the mucosa, with swelling and œdema, especially of the velum and uvula, is associated with the hyperæmia, and the disease culminates at times in chronic relaxation of the velum and elongation of the uvula. The secretion is at first diminished, the patient complaining of "dryness," but later there is an excess of viscid mucus.

In childhood the acute folliculous variety of pharyngitis is very common; that is, the isolated muco-lymphoid glands which are scattered over the posterior wall of the pharynx are especially the centres of inflammatory action.

The patient complains of a constant sense of discomfort, which necessitates frequent acts of deglutition, which are positively painful, although actual swallowing of food is rarely painful except in severe forms of the disease. There is but little systemic derangement in uncomplicated cases.

Diagnosis.—Critical inspection of the pharynx by means of a good light, preferably light reflected from a concave mirror, will establish the diagnosis by

correspondence with the signs above described. In the first twenty-four hours it may be difficult to distinguish simple pharyngitis from the symptomatic pharyngitis of scarlatina, the preliminary pharyngitis of diphtheria, the first stage of acute infectious phlegmon of the pharynx, and pharyngeal erysipelas. The presence of high temperature, perhaps following a distinct chill and accompanied by pronounced systemic derangement, should cause one to anticipate future developments.

Prognosis.—Recovery is hastened by treatment, but in uncomplicated cases it would naturally ensue within ten days. It is supposed that simple pharyngitis may predispose a child to infection by the bacillus diphtheriæ and other pathogenic micro-organisms.

Treatment.—In mild cases a simple gargle of potassium chlorate, ten grains to the ounce, every two hours, is sufficient. This may be made more effective when greater astringency is desired by the addition of tannic acid two grains to the ounce. A variety of other astringents are also available.

In severer cases, especially those which are conjoined with naso-pharyngitis and rhinitis, it is important first to cleanse the entire area by spraying or gargling with an antiseptic alkaline solution :

℞. Sodii boratis
 Sodii bicarbonatis āā gr. xx.
 Ol. eucalypti ℥j.
 Thymol gr. j.
 Menthol gr. ss.
 Ol. gaultheriæ ℥j.
 Glycerini f̄ss.
 Alcoholis f̄3j.
 Aquæ q. s. ad f̄3j.—M.

Sig. Dilute, adding one or two fluidrachms to one fluidounce of water, for use as a spray or gargle.

Young children cannot gargle, and are often terrified by spraying, in which case one may project, through each nostril into the throat, a half-drachm of this diluted mixture by means of an ordinary glass medicine-dropper. After thus cleansing the parts the same astringent gargle may be used; or with larger children and in the hands of the physician, an astringent spray, preferably of the sulphate of iron and ammonium, three to five grains to the ounce, may be applied to the pharynx, and, if need be, by an upward spray-tip to the nasopharynx. The astringents should never be projected through the nose. In painful cases much comfort and some benefit follow spraying by a 1 per cent. solution of cocaine hydrochlorate, and with especially irritable throats its preliminary use will permit subsequent topical applications to be made with greater ease.

When necessary, minute quantities of cocaine may be used in the form of a lozenge, as in the following formula, recommended by Bosworth :

℞. Cocainæ muriatis gr. v.
 Ext. krameriæ gr. ij.
 Sodii bicarbonatis gr. xv.
 Ext. glycyrrhizæ ʒiiss.—M.
 Ft. massa in trochisci No. xxx div.

In office practice as a final spray, or for self-medication, even alone or

following an astringent gargle, we find the following emollient very soothing to highly inflamed mucous surfaces:

R_y. Ol. pini Canadensis ℥ v.
 Ol. eucalypti ℥ ij.
 Ol. gaultheriæ ℥ ij.
 Thymol gr. ss.
 Menthol gr. j.
 "Vaselin oil" q. s. ad f $\frac{3}{4}$ ℥.—M.

Sig. Use with a double-bulb atomizer.

A laxative is usually indicated, even though the bowels may be stated to be regular. Apart from this, little constitutional treatment is required, other than may seem appropriate for any associated conditions.

II. SIMPLE CHRONIC PHARYNGITIS; ELONGATION OF THE UVULA.

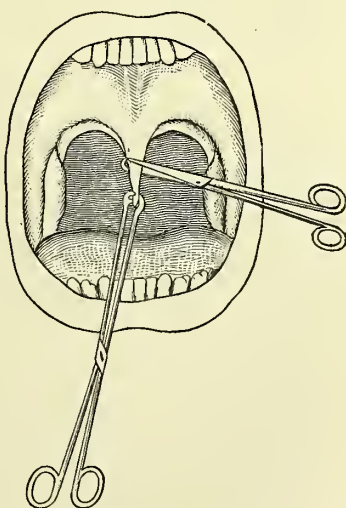
Simple chronic pharyngitis occurs but rarely in childhood, and is then dependent upon diseases of the nose, tonsils, or digestive organs, and the most rational line of treatment, and the only one likely to result successfully, is that indicated by the primary affection. The same is true in part of elongation of the uvula, but only in part, since radical treatment directed to this organ will occasionally be required.

Relaxation of the velum palati and paresis of its muscles are usually associated with lengthening of the uvula, and the disability is due to chronic or recurrent acute inflammation of the nose, naso-pharynx, or pharynx. Frequent necessity to dislodge mucus by "hawking" is somewhat instrumental in its production.

Symptoms.—The chief symptom is a harassing cough, which is found especially annoying on retiring and rising and at times of acute inflammation of the throat. It often causes the child to be treated indefinitely for bronchitis or other invisible disorders, when a critical inspection of the pharynx in a state of quietude would disclose the palate lying on the base of the tongue. Extreme elongation has even served to excite attacks of laryngismus stridulus. Rarely the uvula is bifid, a congenital defect which predisposes it to elongation.

Treatment.—Concerning the treatment, palliation may be secured, even cure in recent cases, by an astringent spray or gargle. More often this will fail to produce wholly satisfactory results, and then attention must be given to whatever abnormality underlies the elongation of the uvula; if the tonsils be hypertrophied, they should be abscised; if there be naso-pharyngeal adenoid hypertrophy, it should be removed, etc. If the difficulty then continue, one should not hesitate to abscise the surplus portion of the uvula, leaving it of normal length. It is most quickly done by a tonsillotome, but can readily be accomplished by forceps and scissors. (Fig. 1.)

FIG. 1.



Abscission of the Uvula.

III. CHRONIC FOLLICULOUS PHARYNGITIS.

Although, as previously stated, simple chronic pharyngitis occurs but rarely in childhood, chronic folliculous pharyngitis is not uncommon. It is characterized by enlargement of the isolated muco-lymphoid follicles which are scattered over the posterior wall of the pharynx and arranged in a chain in each lateral angle of the throat behind the posterior pillar. These are single follicles of the same histological structure as the tonsils, which are compound glands. It is natural, therefore, that they should become hypertrophied in response to the same underlying dyscrasia—lymphatism—which predisposes patients to hypertrophy of the tonsils and of the naso-pharyngeal adenoid tissue. Indeed, in children the disease is usually conjoined with the latter pathological states. Symptoms are manifest only in pronounced cases, and then, usually, at times of an acute exacerbation. A constant tendency to “hawk,” a sense of discomfort, and, in rare instances, a sense of a foreign body in the throat, are the most important. On inspection one observes small round eminences dotted irregularly over the posterior wall of the pharynx, and ridges of reddish hue in the lateral angles. The latter aspect of the disease, when especially marked, has been designated, in recent works, pharyngitis lateralis.

Treatment.—The enlarged follicles should be destroyed by touching each one with the galvano-cautery point-electrode. Three or four may be cauterized at each sitting, and several sittings will be required. The result is very satisfactory. When tonsillar and naso-pharyngeal adenoid hypertrophy is also present, this condition should first be removed, in which case further treatment often becomes unnecessary.

IV. ACUTE FOLLICULOUS TONSILLITIS.

The infectious nature of most cases of folliculous tonsillitis is now definitely established; yet other cases are, seemingly, of simple catarrhal origin, devoid of pathogenic germ infection; it is therefore evident that one can distinguish, and should describe, at least two forms of this disease: infectious pseudo-membranous tonsillitis and simple folliculous tonsillitis.

INFECTIOUS PSEUDO-MEMBRANOUS TONSILLITIS

is also termed “croupous tonsillitis,” “tonsillitis lacunaris,” “diphtheritic sore throat,” and “pseudo-diphtheria,” although the latter term has been indiscriminately applied also to scarlatinous diphtheria and to all forms of membranous pharyngitis not caused by the Klebs-Loeffler bacillus.

Etiology.—The infectious nature of certain forms of acute folliculous tonsillitis has long been suspected, yet the fact has not been generally credited, for the reason that when the clinical evidence of infectiousness was conclusively present the disease would be attributed to diphtheritic origin or the subject be dismissed as a mere coincidence. We now know that the true *bacillus diphtherie* is not present in this disease, but that the form described under the name of infectious pseudo-membranous tonsillitis, or croupous tonsillitis, is caused by local infection by any one of several species of pathogenic micro-organisms; *e. g.* streptococcus erysipelatosus, streptococcus pyogenes, staphylococcus pyogenes aureus, staphylococcus albus, etc.

Symptoms.—Infectious pseudo-membranous tonsillitis is characterized by deep congestion, but often only by moderate swelling of the tonsils and by a punctated exudate of pseudo-membrane, the spots of which are in size from 2 to 4 mm. in diameter, and are attached around the follicular openings, pre-

senting the appearance as if the crypts were also lined by the same material; unlike the cheesy pellet, the exudate in its typical form is thin, translucent, and intimately connected with the underlying mucosa. Two or more puncta may join at their borders and form larger spots, but after cleansing away all mucopurulent matter this punctated conformation of even the larger areas may be readily discovered (Fig. 2). In addition to the tonsils, any or all of the muco-lymphoid glands in the pharynx may be likewise affected, especially the chain of glands located just behind the tonsil and separated from it by the posterior pillar; but the pseudo-membranous exudate is limited absolutely to the glandular structures of the pharynx, although careful cleansing and critical inspection will be required to demonstrate this fact.

The attack is ushered in by chilly sensations, perhaps preceded, for a day or so, by malaise, and followed by a temperature of 102° to 105° F., with consequent febrile symptoms. After one to three days the temperature falls materially; the pain, which has been quite severe, gradually ameliorates, and within one week convalescence is established. The cervical lymphatic glands are often secondarily infected, as evidenced by swelling and tenderness, which last for two or three weeks. Suppurative cervical adenitis and cellulitis may follow in rare instances. Transient albuminuria is an occasional complication.

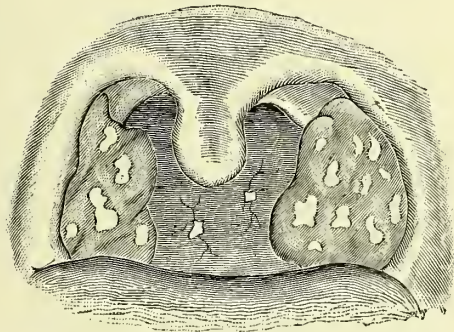
Diagnosis.—The opinion of bacteriologists that in these affections diphtheria can only be excluded positively by the absence of the Klebs-Loeffer bacillus, as determined micro-

scopically, is doubtless correct as applied to rare border-line cases; but commonly a differential diagnosis can also be made with greater promptness and with reasonable certainty from the macroscopic signs and clinical symptoms.

True diphtheritic exudation may commence at the orifices of the crypts of the tonsil, but does not long remain limited to the tonsils and muco-lymphoid glands of the pharynx, as does the exudate of tonsillitis. The diphtheritic membranes will extend within twenty-four hours to the pillars, velum, or pharyngeal wall. The exudate of tonsillitis is thin, and not materially raised above the surface; it is white, translucent, and presents a living, clean aspect devoid of necrotic change; while the exudate of diphtheria is thickish or protruding from the surface, opaque, and dirty-yellow or rapidly becoming so—appearances indicative of necrotic change.

The exudate of tonsillitis is punctated, the spots corresponding to the follicular openings, and, while two or more puncta may join at their borders and form larger areas, after careful cleansing, critical inspection, under thorough illumination, will disclose this punctated conformation, which distinctly differs from the diffuse plaque of diphtheria, even when, for the time being, the diphtheritic exudate occupies the tonsil alone.

FIG. 2.



Acute Infectious Pseudo-membranous Tonsillitis (follicular). The two whitish points on the posterior wall represent exudate formed on isolated muco-lymphoid follicles.

SIMPLE FOLLICULOUS TONSILLITIS.

With the simple form there may or may not have been previous chronic

hypertrophy or inflammation: it is conditioned, if not caused, by "taking cold," *i. e.* by refrigeration of some part of the body surface, which determines vascular engorgement of the tonsils, exactly as in another individual it may occasion vascular engorgement of the nasal turbinated bodies. The tonsil swells, the follicular openings are obliterated and the pent-up secretion acts as a further irritant; it becomes inspissated and mixed with epithelial débris; it is soon forced out to the surface of the gland in the form of "cheesy" pellets, which are altogether different from a pseudo-membrane, and which protrude from the narrowed follicular openings. Finally, when the tonsils are free of this accumulated débris, or at times earlier if the globules are forcibly dislodged and removed, the tonsillitis rapidly subsides. It is not usually preceded by a distinct chill, and not accompanied by much fever or systemic depression. It is without evidence of primary parasitic infection as a cause, and therefore not contagious; it is capable, however, of being transposed into a conglomerate variety of tonsillitis by secondary infection with pathogenic micro-organisms, thus becoming contagious.

In fact, between these two types of tonsillitis are observed numerous cases of mixed variety which present all degrees of approximation to one or the other type.

General Treatment.—The rheumatic diathesis is frequently associated directly or indirectly with tonsillitis, in which case salicylate of sodium or salol should be administered internally. Otherwise, the tincture of the chloride of iron, 1 part to 10 parts of glycerin, may be administered every hour without further dilution in the dose proportionate to the age of the child, both for its local effect, as it is diffused over the fauces in swallowing, and for its systemic influence.

For the high febrile action of the first day or two we have been accustomed to give minute doses of tincture of aconite, conjoined with potassium bromide, disguised in solution by a few minims of spirit of peppermint, and to which may be added very small quantities of morphine if it is needed to control pain.

Of late years, antipyrine or phenacetin has been often substituted advantageously for the aconite and bromide mixture. A saline laxative is nearly always needed.

Local sprays by a hand-atomizer are of the greatest benefit when the child is old enough to tolerate them. An alkaline and antiseptic lotion (See Acute Pharyngitis) is to be preferred. This should be sprayed every three hours through the mouth, and also through the nose, into the naso-pharynx, thus cleansing that cavity, as well as the fauces, of the viscid muco-purulent matter which accumulates and conduces to much discomfort.

Hydrogen peroxide, diluted to the point of freedom from production of smarting sensation, is also an excellent local spray, especially if used alternately with the one above mentioned; and either or both of these may be used following a preliminary spray of 1 per cent. solution of cocaine hydrochlorate, which serves to control pain and super-irritability of the fauces.

Generally speaking, it is best to avoid the use of cotton swabs and brushes. Gargles may be substituted for sprays when necessary, or made to supplement spraying, and for use as a gargle the formula for spray above referred to should be diluted doubly as much as for use in a spray.

With very young children the naso-pharynx and fauces can be readily cleansed by the same solution freely diluted, warmed, and injected gently in small quantities by a small syringe or an ordinary medicine-dropper through the nares.

V. PERITONSILLAR ABSCESS, OR SUPPURATIVE TONSILLITIS.

This condition is also termed acute parenchymatous tonsillitis, phlegmonous tonsillitis, quinsy, etc., but of these terms the best is peritonsillar abscess, because it is descriptive, since the suppuration does not occur in the tonsil itself, but in the cellular tissue around it or above, behind, in front, or to the outer side of the gland. The disease is comparatively rare in early childhood, but about 3 per cent. of all cases occur under ten years, and about 6 per cent. under fifteen years, of age.

Etiology.—The direct cause of suppuration here, as elsewhere, is infection by specific pathogenic micro-organisms from some source, either from within or without the body.

The predisposing causes are exposure, the rheumatic diathesis, chronic tonsillitis, and acute folliculous tonsillitis.

Symptoms.—A chill or chilly sensation is followed by a temperature of 102° to 105° F., and consequent febrile symptoms. About the same time a sense of soreness and fullness is perceived in one side of the throat, followed by lancinating pains which dart through to the ear, and, later, by a deep-seated throbbing pain as suppuration ensues. On inspection the swelling is seen to extend to the median line of the throat, and even far beyond, in severe cases projecting upward into the naso-pharynx and downward along the side of the pharynx, sometimes leaving only the smallest chink available for respiration and deglutition. The latter function is painful, and the diet must be confined to liquids, for the reason, also, that the lower jaw is "set" so that the mouth can be opened only about half an inch.

Viscid mucus accumulates in the partially occluded pharynx and in the naso-pharynx, causing suffocative attacks and necessitating painful efforts to clear the throat. Indeed, for a night or two the patient cannot assume a recumbent position or sleep uninterruptedly, as voluntary efforts are required to maintain patency of the throat. The uvula becomes œdematous, and the opposite tonsil is usually somewhat swollen, often suppurating later, although simultaneous suppuration of the two sides is rare.

Diagnosis.—During the first twenty-four hours the disease cannot be distinguished with certainty from folliculous tonsillitis, which, indeed, often precedes the peritonsillar abscess. Later, the diagnosis is established by the characteristic distortion of the throat, as represented in Fig. 3, in which it is seen that the tonsil itself is not the chief seat of swelling, but that this gland is projected inward by tumefaction in the cellular tissue of the velum palati.

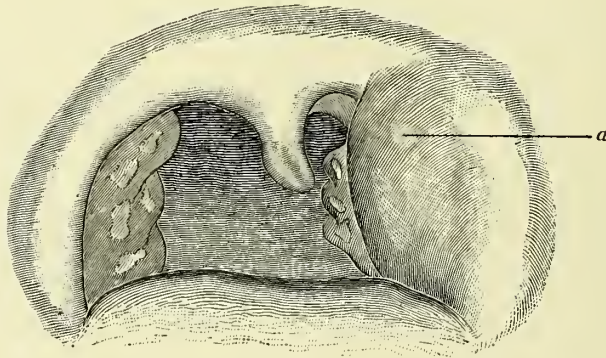
Prognosis.—This is favorable, except in cases of rare complications, such as œdema of the larynx, extensive burrowing of pus, or hæmorrhage.

Treatment.—During the first twenty-four or thirty-six hours an effort should be made to abort the disease, and to this end the internal and local medicinal treatment is much the same as that described for folliculous tonsillitis—a saline laxative, the immediate administration of salicylate of sodium in full doses because of the common dependence of the disease on the uric-acid diathesis, and tincture of aconite with potassium bromide as an adjuvant. The same alkaline and antiseptic spray which is recommended for folliculous tonsillitis should be used every hour or half-hour, and in the same manner, spraying through the mouth, and to a less extent through the nose. In the early stage of the affection the application of cold externally by means of Leiter's coil would assist in aborting the suppurative inflammation were it as feasible with restless children as with adults. As soon as it becomes evident that sup-

puration must occur, a hot poultice, applied externally over the corresponding part of the neck, will both ease the pain and hasten the formation of pus.

At the earliest moment that pus is indicated with reasonable certainty by fluctuation or an effort at "pointing" the abscess should be punctured, preferably by a long, slightly curved, double-edged bistoury devised for the purpose, or, in the absence of this instrument, by an ordinary sharp-pointed bistoury. The puncture should not be made into or through the tonsil itself, but somewhat above and to the outer side of the gland into the anterior surface of the velum, where the pus actually is located, in the cellular tissue of the velum palati and palato-glossal fold (Fig. 3).

FIG. 3.

Peritonsillar Abscess: *a*, point for puncture.

VI. HYPERTROPHY OF THE TONSILS.

The exact function and size of normal tonsils are questions of interest which are answerable only in a general way. Histologically, they possess the structure of both a lymphatic and a mucous gland, and, anatomically, they are in close connection by lymph-channels with the cervical lymphatic glands. The inference is that they are lymphatic glands, possessing the function of similar glands elsewhere located, which by virtue of their position in the fauces have been endowed also with mucous elements for lubricating purposes. The natural size approximates that of an almond-kernel.

Etiology and Pathology.—The predisposing cause of enlargement of the tonsils is a peculiar diathesis now termed "lymphatism," the local manifestations of which include also enlargement of the naso-pharyngeal tonsil, or "adenoids," and of the muco-lymphoid glands of the pharynx and base of the tongue. This diathesis is certainly not identical with scrofula, even in the limited sense to which that term is now restricted, for lymphatism frequently manifests itself in children who are otherwise robust, yet the condition seems allied to, and often conjoined with, scrofula. Climatic inequalities furnish adequate exciting causes.

In the usual form of the disease, that of mere hypertrophy, there is simply an overgrowth, both in size and number, of all the natural elements of the gland—the lymphoid bodies, crypts and follicles, mucous glands, and connective tissue.

Another variety of hypertrophy of the tonsils, named by Bosworth the hyperplastic form, which is rare in children, but common in adults, results from repeated attacks of acute inflammation and consists chiefly of hyperplasia of

the fibrous connective-tissue element, with a less degree of enlargement and multiplication of the lymphoid bodies. Such tonsils are dense and fibrous, while those of the first type are soft and friable. Between these two types, exist all degrees of variation, both in contour and texture.

Symptoms.—Moderate enlargement only will occasion a tendency to recurrent attacks of acute tonsillitis, and any degree of hypertrophy unquestionably predisposes the child to diphtheritic infection and increases the gravity of the latter disease when it occurs.

The effects of mechanical obstruction to respiration occasioned by enlarged tonsils, either alone or especially in conjunction with enlargement of the nasopharyngeal tonsil, will be described in the article on Naso-pharyngeal Adenoid Hypertrophy, and I need only mention here the more prominent features.

Mouth-breathing can be caused even by enlarged faucial tonsils alone—by their projection backward and upward into the pharynx in such a way as to interfere with the passage of air inspired through the nose. Mouth-breathing in turn causes deformed development of the facial bones and muscles and an idiotic expression of countenance and mental stupidity; also, deformed development of the chest and thoracic weakness. The recumbent position and absence of voluntary muscular control to keep the throat open aggravate the obstruction to both nasal and oral respiration at night, so that the patient is frequently awakened or thrown into a nightmare by a sense of dyspnoea. Deglutition and mastication are impaired in proportion to the extent of the disease, although it is probable that deficient oxygenation of the blood and disturbed rest at night, together with subsequent thoracic deformity, are the chief factors in seriously stunting the development of the child.

Treatment.—Abscission is the only satisfactory method of treatment when the enlargement is sufficient to occasion the symptoms of mechanical obstruction.

It is probable that the syrup of iodide of iron so far tends to correct the underlying constitutional dyscrasia as to prevent recurrence after operation, and even to cause partial reabsorption of very slight and recent overgrowths; but we have never been able to discern therefrom any permanent reduction of tonsils which were greatly or even moderately enlarged. Local astringents are wholly inadequate. Ignipuncture or galvano-cautery puncture affords only palliation for the milder cases. We have repeatedly found it necessary to abscise tonsils after months had been spent with this somewhat painful and ineffective mode of treatment.

The wire snare is an excellent means of abscission when the child is anæsthetized, as when combining this operation with that for “adenoids;” but otherwise it is slow and painful, and, like the galvano-cautery snare, it requires more time and quietude for adjustment than are available with young children when not anæsthetized. An anæsthetic is not usually necessary when the faucial tonsils alone are to be abscised, although it is decidedly best to administer ether when the combined operation for removal of the faucial tonsils and naso-pharyngeal “adenoids” is to be made. Also, with unusually excitable or obstreperous children ether may be administered.

The tonsillotome is still the best implement for children who are not anæsthetized, because of the rapidity, precision, and comparative ease with which this method can be practised. With older children it is best to use a preliminary spray of 5 per cent. cocaine solution. Younger children are apt to be terrified by spraying, and it is best to omit it. The pain is not really great.

The Mathieu tonsillotome is well adapted to the purpose, especially for children, and it is the one now in general use. The mechanism is very

ingenious, being fitted with a fork attachment which is designed to transfix the tonsil, and withdraw it from its bed by the same motion of the operator's fingers which draws the ring-knife home. The much-vaunted Mackenzie tonsillotome is an unnecessarily cumbersome instrument.

The author has described elsewhere a simplified instrument which he has

FIG. 4.

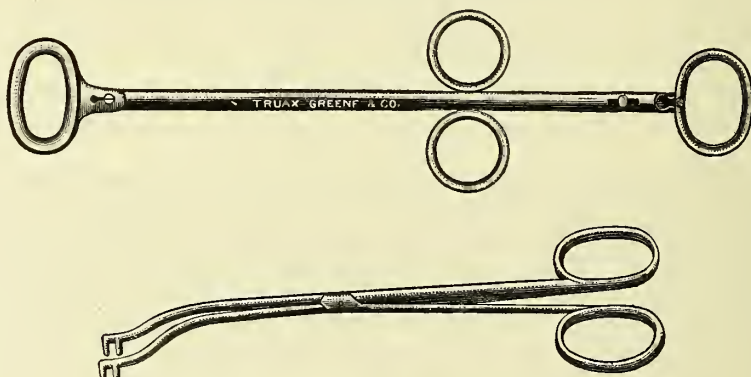


Mathieu's Tonsillotome.

used for years with the utmost satisfaction. It is the Mathieu guillotine, so constructed as to do away with the fork attachment (Fig. 5).

In place of the fork he uses, held in the other hand, a specially constructed vulsellum (Fig. 5), by which the tonsil can be grasped, drawn out of its bed, and abscised at the point desired with much greater accuracy than by the fork attachment (Fig. 6). He has found the action of the fork to be largely accidental, dependent on the size and shape of the tonsil and the amount of gagging by the patient—that now it determines too deep an

FIG. 5.



The Author's Tonsillotome and Tonsil Vulsellum.

abscission, and, again, misses the tonsil entirely, especially if this happens to be rather small or flat. In other words, the new instrument, assisted by the vulsellum, will abscise many tonsils that could not be satisfactorily grasped by the old mechanism, and it will abscise all tonsils with a reasonable degree of accuracy at the proper line.

One can also by this instrument more easily avoid wounding the anterior and posterior pillars, which eliminates one of the sources of persistent hæmorrhage. The instrument therefore conduces to safety by virtue of greater possible precision in operating. It is less formidable in appearance and is easy to use. No tongue-depressor is necessary, the body of the tonsillotome answering this purpose, at the same time that the vulsellum prongs grasp the tonsil to draw it from its bed into the ring of the tonsillotome.

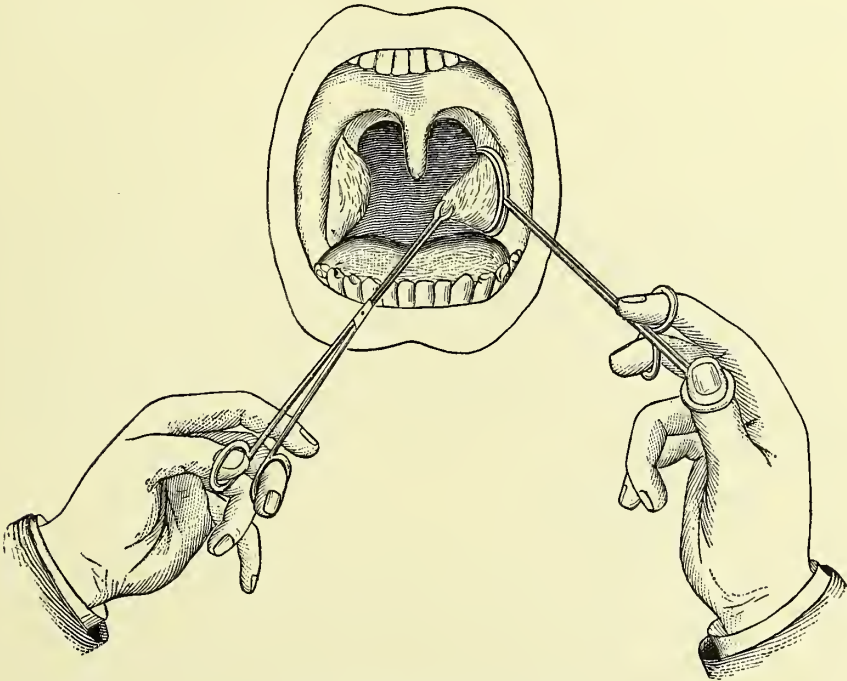
The proper line or point for abscission I believe to be close to the base of the gland, but not so close as to constitute a total extirpation. A stump should be left, but one not much larger than the normal gland, and not of suf-

ficient size to protrude from or widely separate the pillars of the fauces. A total extirpation would seem unnecessarily hazardous on account of difficulty of access to bleeding vessels should hæmorrhage occur, and I cannot think that hæmorrhage is any less prone to occur after total extirpation, as recently stated, than after abscission.

On the other hand, when a considerable portion of the gland is left, only the cortical layer being removed, redevelopment of the growth is common.

Very large and densely fibrous tonsils in older children are best removed

FIG. 6.



The Author's Method of Tonsillotomy.

by the galvano-cautery snare, since they are especially apt to bleed if cut, and are difficult to abscise by a cold wire. In rare instances hæmorrhage even then occurs, either primarily when the wire is overheated, or secondarily on the separation of the slough. The chief objection to the method for general use is the intense inflammation of the fauces which is liable to follow it. This can be, in part, but not wholly, obviated if one is careful not to singe the pillars, which, however, are not so easily avoided in the use of the cautery snare. To this end, Dr. Jonathan Wright has adapted the frame of the Mackenzie tonsillotome to galvano-cautery purposes by substituting for the steel blade a wire mounted on compressed paper and to be connected with a battery.

Consideration of this subject would not be complete without reference to the views of Dr. Harrison Allen of Philadelphia, as advanced in a recent essay before the American Laryngological Association. He believes "that abscission should be restricted to the removal of the superficial or cortical part of the tonsil, and in preference to the treatment by amputation of the whole mass; that after removal of such cortex, should the crypts be closed, he would search

for hidden canals, and when found pass a probe or director through them and freely divide the overlying tissues, incising thus the tonsil in any direction and to any required depth. After this is done the separate coarse lobules can be severally taken up by forceps and removed, care being taken to avoid touching enveloping folds of mucous membrane." It is evident that this would be an impossible method with most young children, because of tediousness, but it may be advantageously utilized with older patients.

The only serious objection to abscission of the tonsils is the rare possibility of troublesome hæmorrhage, which has seemed to a few extreme conservatives to justify avoidance of the operation; but a greater risk is assumed in every phase of life, in travel, and in pursuit of business and pleasure. It is stated that Elsberg made the operation eleven thousand times with but two cases of even alarming hæmorrhage, and Morell Mackenzie, whose experience must have been enormous, only once met with a case in which the bleeding appeared actually to endanger life. Only one authentic case of death of a child from hæmorrhage after tonsillotomy is recorded in modern literature, and it is probable that this case need not have ended fatally but for a deception of the operator relative to the seriousness of the hæmorrhage, by reason of the blood being swallowed by the young child and not expectorated; which caused the adoption of a less vigorous treatment than otherwise would have been used.

When one considers the number of cases, beyond computation, of tonsillotomy in children, and the few reported cases of hæmorrhage, *one must regard it as among the safest of even minor operations.*

The treatment of severe hæmorrhage may consist, first, of a trial of the astringents and styptics. The most popular of these is Mackenzie's mixture of tannic and gallic acids:

R. Acidi tannici 3vj.
 Acidi gallici 3ij.
 Aquæ f5j.—M.

Sig. Sip and swallow half-teaspoonful quantities at short intervals.

If this fail, it is probable that any simple astringent or vaso-contractor will fail.

Ice, held in the mouth and swallowed, is also an efficient remedy.

Pressure may be successfully applied by grasping the tonsil firmly between the thumb, held within the mouth and enveloped in three or four layers of linen, and the fingers held over the corresponding part of the neck. It must be maintained sometimes for an hour or more.

When the simpler expedients fail, then the bleeding points and surfaces should be accurately located and thoroughly seared by the actual cautery, or the galvano-cautery if at hand. For this purpose one needs several small sponges mounted on long sponge-holders, which, if not at hand, may be substituted by wooden sticks (sponges are much more effective than absorbent cotton); also, a small surgical retractor, like a tracheotomy retractor, in the absence of which a palate hook, or even a bent probe, will serve. An assistant is desirable to hand and clean the sponges.

Under the illumination of a head reflector, the throat should first be well sprayed with a 5 per cent. solution of cocaine, and sponged clear of clotted blood; the bleeding surface can then be exposed to view by holding aside the anterior pillar by means of the retractor, when by rapid sponging the bleeding points can be discerned and then cauterized.

As a substitute for the galvano-cautery one may use a thick wire heated

to redness over a gas-flame. We have used this means successfully with adults, but have never had occasion to apply it with children. If necessary, however, we would endeavor to do so with young children by first administering chloroform and inserting a Whitehead gag, as in operation for cleft palate, placing the patient with the shoulders elevated and the head pendent, so that blood could not gravitate into the trachea.

When the hæmorrhage is comparatively slight exact cauterization of the bleeding points by solid nitrate of silver is effective. Torsion is applicable only when a spurting artery can be seen.

As a last resort, may be mentioned ligation of the external carotid artery, as advised by Delavan, in preference to ligation of the common carotid, which latter might permit hæmorrhage to continue by collateral circulation through the circle of Willis.

VII. RETRO-PHARYNGEAL ABSCESS (RETRO-PHARYNGEAL LYMPH-ADENITIS).

It is now well established that retro-pharyngeal abscess arises ordinarily not in caries of the cervical vertebræ, but in suppurative inflammation of the lymphatic glands which are imbedded in the posterior pharyngeal wall. In harmony with accepted views of the origin of pus elsewhere, the source of this inflammation must be infection, either from within or without the body, by some one or more of the pathogenic micro-organisms which produce supuration.

Children are especially prone to inflammations of the lymphatic system. Cervical lymphadenitis is common among them. Frequently it is tuberculous, but often it is not, and usually the acute suppurative variety results from infection by a previously existing tonsillitis. So also with retro-pharyngeal abscess: it is most reasonable to regard it as a secondary infection of the pharyngeal lymphatics from inflammation of exposed and associated muco-lymphoid glands, like the faucial and naso-pharyngeal tonsils. But, whatever the source of infection, whether primary or secondary, the initial stage of retro-pharyngeal abscess is retro-pharyngeal lymphadenitis. Moreover, the lymphadenitis may be of a non-suppurative type, or the disease become arrested in this stage, undergoing resolution without the formation of an abscess.

Bokai reports a case of retro-pharyngeal lymphadenitis in a child eight months old, in which tracheotomy was necessitated by the supervention of alarming symptoms of suffocation. The posterior wall of the pharynx showed diffuse hard swelling without fluctuation, and a deep incision into the mass had yielded no pus. After the tracheotomy resolution was quickly established. This simple lymphadenitis has been but rarely observed in this country, but Bokai, in addition to 400 cases of abscess, mentions 112 cases of simple retro-pharyngeal lymphadenitis as having passed under his observation in the Pester Kinderspital. (See note at end of this chapter.)

In rare instances the source of infection may be rhinitis, communicated through the nasal lymph-channels, or, still more rarely, a suppurative otitis; but, as previously intimated, folliculous and suppurative forms of tonsillitis, as well as those forms of tonsillitis and pharyngitis which are symptomatic of the exanthemata, may reasonably be regarded as the most frequent causes of retro-pharyngeal lymphadenitis, which in turn may proceed to the formation of an abscess. Cases which originate in any of these ways are grouped by Bokai under the term "idiopathic;" and of 204 cases analyzed, he placed 189 in this class, in contradistinction to only 7 cases secondary to caries of the vertebræ,

7 cases from burrowing of pus from abscess in the neck, and 1 case of traumatic origin.

Symptoms.—The disease may commence quite insidiously or it may culminate rapidly. Attention is directed to the throat by a deep-seated pain, dysphagia, and, later, by dyspnoea. When located low down in the laryngopharynx, a comparatively small abscess may speedily occasion suffocative symptoms. Critical inspection or palpation of the throat will disclose a swelling of the posterior pharyngeal wall, which may be either in the median line or somewhat to one side.

Diagnosis.—The disease is distinguished from oedema of the glottis by inspection, which reveals pharyngeal instead of laryngeal swelling, and from both diphtheritic and spasmodic laryngitis in the same manner; moreover, in both forms of croup the voice is impaired, which is not the case in retro-pharyngeal abscess.

Prognosis.—The affection usually terminates in recovery in from five to fifteen days, the abscess discharging spontaneously in many instances. In a considerable proportion of cases, however, prompt recognition of the disease and evacuation of the pus is necessary to avert a rapidly-fatal issue by suffocation, or, in rarer cases, to prevent burrowing of the pus into the oesophagus, larynx, mediastinum, or pleural cavity.

Treatment.—As soon as pus has formed it should be evacuated by making an incision as near the median line as possible, and then the head of the child should be inclined well forward to prevent the pus from running into the larynx. An ordinary bistoury will suffice for the incision. An exploratory puncture may be made at any time to determine the presence of pus. In Bokai's experience tracheotomy has been but rarely necessary, but it should be promptly performed if puncture of the swelling does not relieve the suffocative symptoms by evacuation of pus.

The syrup of iodide of iron and nutritive tonics are indicated.

VIII. NASO-PHARYNGEAL ADENOID HYPERTROPHY.

This disease, which is variously known as "adenoid hypertrophy in the naso-pharynx," "adenoid vegetations," and "third tonsil," in multiplicity of cases and gravity of consequences will bear comparison with any other affection of the upper respiratory tract. In the normal state isolated and aggregated muco-lymphoid follicles of the same adenoid structure as those in the pharynx are imbedded throughout in the mucous and submucous tissues of the naso-pharynx. Histologically, each in its simplest form consists of a depression of the mucous membrane lined with its epithelium and enveloped in a stratum of reticular connective tissue, entangled in which are numerous lymphoid cells, lymphoid bodies (closed follicles), and lymphatic and other vessels. Morphologically, they are closely related to the faucial tonsils, which are compound aggregations of the same. At the vault of the pharynx a number of these follicles are grouped together, forming a compound gland analogous to the tonsils, and known as the third tonsil, the pharyngeal tonsil, or the tonsil of Luschka. In the normal state this is not of sufficient size to deserve such appellation, but when hypertrophied, as it frequently is, it bears some resemblance to the faucial tonsil in a state of enlargement. Several sorts of aggregation are distinguishable clinically by rhinoscopic inspection. Of these the more common are: (1) the *fimbriated variety*, in which the growth is composed of several cock's-comb-like masses closely packed together; (2) the *stalactitic form*, in which multiple pear-shaped bodies are pendent, like stalactites, from

the vault of the pharynx, and to which the name "adenoid vegetations" is most truly applicable; (3) the *individuate variety*, in which the mass is made up, in large part, of but a single neoplasm, of firmer consistency, smoother surface, and more or less irregular contour according to size and degree of impaction.

Regarding consistency, this is found to vary in accordance with the amount of fibrous tissue in its composition. In the fimbriated and stalactitic forms the adenoid element predominates, rendering them friable and soft to the touch, while the individuate variety often contains much fibrous tissue, which gives it greater density and tenacity. Between these forms are encountered all degrees of variation both in contour and texture.

Etiology.—Children of syphilitic and tuberculous parents and those otherwise the victims of scrofulosis are predisposed to it, but children in other respects robust are also affected.

The term "lymphatism" has been introduced as a recognition of an underlying dyscrasia which is characterized by hyperplasia of this and other mucolymphoid structures, including the faucial tonsils. Climatic inequalities furnish adequate exciting causes.

Symptoms.—The space of the naso-pharynx is designed to serve as a common area of air-communication between the five openings which enter it. The Eustachian tubes open into it, one on each lateral wall posterior to the nasal choanæ, and upon perfect patency of these openings, together with free nasal respiration, the power of hearing is dependent; for ventilation, with normal air-pressure in the cavity of the middle ear, is essential to correct auditory sense. The adenoid excrescences, when large, are forcibly compressed between the lateral walls of the naso-pharynx or they overlap the tuber of the Eustachian orifice from above, acting in either case as a stopper to one or both openings; or else the vegetations which are crowded in above and behind the Eustachian tubes deform and close the orifice by forcing its upper projecting lip downward to meet the lower border of the rim. Fig. 7, accurately drawn from nature, is a typical representation of an average case, in which the naso-pharynx is seen to be occupied by a fimbriated adenoid mass which occludes, in large part, the posterior nasal choanæ, and so presses downward the upper lip of the tuber of the left Eustachian orifice as to practically close the channel to the middle ear.

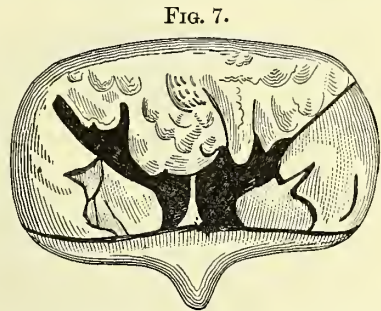


FIG. 7.

Naso-pharyngeal Obstruction by Adenoids.

Again, even with lesser hypertrophy, the accompanying catarrhal state is prone to extend by continuity of surface along the Eustachian tube, and to excite exudation or suppurative inflammation of the middle ear. Deafness, therefore, is frequently a deplorable symptom, and one which is liable to become permanent unless speedy relief be afforded.

Into this space open also the posterior nares, the natural respiratory passage being *viâ* the nose and naso-pharynx. Adenoid hypertrophy, therefore, serves as a plug to the posterior nasal openings, and obstructs nasal respiration completely or in part according to the degree of glandular enlargement. From this point we find it a matter of exceeding interest to trace the origin and development of each successive step in the series of deformities consequent upon this condition. The plugging up of the posterior nares necessitates oral breathing, and the constantly open mouth interferes with the normal adaptation of

certain facial muscles, which in turn effects radical changes in the contour of the soft and developing bones of the face, the whole resulting in a physiognomy characterized by a vacant, stupid, almost idiotic expression of countenance, which can be better illustrated by a photograph from nature than described (Fig. 8).

FIG. 8.



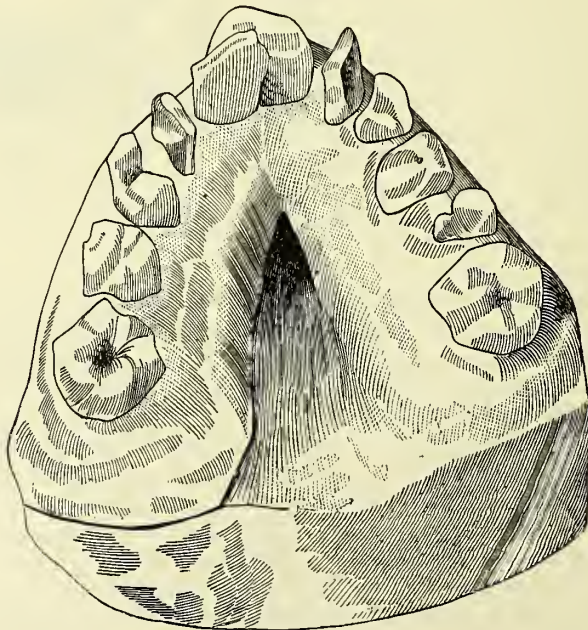
Characteristic Physiognomy of Adenoid Vegetations (from a photograph by the author).

The hanging lower jaw causes the face to appear elongated. The nose is pinched or its alæ distended, while the angles of the mouth and eyes have a drawn appearance.

Moreover, the air-cavities in communication with the nose, as the frontal, maxillary, sphenoidal, and ethmoidal sinuses, which are essential to the proper expansion of their respective bones, cease to develop when the circulation of air through the nose is interfered with, thus altering nature's intent regarding the dimensions of the face and head, and still further deforming the physiognomy. Augmentation of atmospheric pressure upon the buccal surface of the palate process, and the impact of air-currents to and fro during mouth-breathing, together with the diminution of intra-nasal air-pressure incident to nasal obstruction, gradually force upward the centre of the hard palate, and

change thus the obtusely rounded Romanesque arch into one of Gothic shape—the pointed or high-arched palate commonly existing in association with long-

FIG. 9.



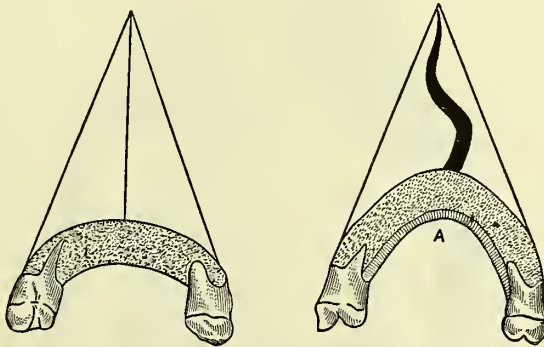
High-arched Palate.

continued and excessive adenoid development during childhood (Fig. 9). Elevation of the palatal arch lessens the traverse diameter of the jaw, and causes it to grow pointed in front—the so-called V-shaped indenture; and with the resulting contraction of the alveolar process, the teeth, especially those near the point, are crowded into various grotesque aggregations or are rotated on their axes—a condition depicted in Fig. 9, drawn from a typical case, in which the two central incisors overlap, and the two lateral incisors undergo a quarter rotation and stand at right angles to the alveolar process.

It is proper to state that this relation of mouth-breathing to deformed indentures is questioned by some dental authorities, who attribute the elevation of the palatal arch solely to a perverted production of the permanent teeth. The association between the adenoid hypertrophy as a cause of mouth-breathing and the high-arched palate is, however, so constant that an etiological relationship is most probable.

Next, elevation of the palatal arch must produce contortion within the nose, for the septum, composed of the vomer, the perpendicular plate of the ethmoid bone, and its cartilaginous portion, is unequal in power of resisting compression to the bones by which it is incased. Designed by nature to fill vertically the

FIG. 10.



natural space between the roof of the nose and its floor, the abbreviation of this space by elevation of the palatal arch through the instrumentality of nasopharyngeal adenoid hypertrophy cannot result otherwise than in forcing the septum to provide for itself by bending and curving laterally in various directions—a condition which is diagrammatically represented in Fig. 10.

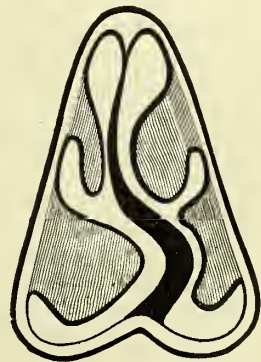
The septal deflection acts as an additional impediment to nasal respiration and drainage, and becomes a potent factor in the evolution of hypertrophic rhinitis or that form of nasal catarrh characterized by enlargement of the turbinated bodies (Fig. 11).

Headache is also complained of, although a sense of mental obtundity and heaviness is more usual than absolute pain in the head.

Finally, not only, as before said, do these unfortunates *look* stupid, but they really *are* stupid, and exhibit abundant evidence of mental hebetude, with inability to fix the attention, to learn, to memorize, or to reason.

Three varieties of thoracic deformity are observed to accompany obstruc-

FIG. 11.



tive naso-pharyngeal adenoid hypertrophy, the association of one or other form, in advanced cases, being so constant that a direct causal relationship, although difficult of absolute demonstration, can reasonably be assumed.

For the induction, however, of two of these forms, the "pigeon-breast" deformity and the "barrel-shaped" chest, the intermediation of still another symptom, bronchitis, seems essential; but adenoid hypertrophy is an etiological factor in the production of chronic bronchitis. Especially in neurasthenic individuals it is exquisitely sensitive to reflex-producing impressions, and its irritation may result, reflexly, in spasm of the glottis, cough, asthma, and parietic vaso-motor bronchitis.

The third variety of thoracic deformity, the "flat chest," is due directly to obstruction by the adenoid growth itself, and is an indrawing of the chest-walls, especially a shortening of the antero-posterior diameter, which results from an insufficient air-supply to the lungs. The chest becomes flat and thin (Fig. 12), has a sunken appearance over the lower part of the sternum, perhaps a deep concavity at the ensiform cartilage, with depressed intercostal spaces.

FIG. 12.

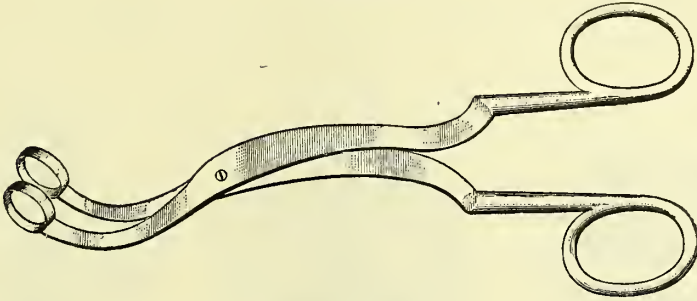


Flat-chest Deformity (Hooper).

Rachitis, so often associated with depraved nutrition, is doubtless the predisposing condition to all of these forms of chest deformity.

Treatment.—For pronounced hypertrophy the only satisfactory method of treatment is removal by surgical means. Many methods by cautery, snare, curette, and forceps, without general anæsthesia, have been described. With older children it makes little difference which of these methods is employed, so that the object is thoroughly accomplished. With young children, however, who will not hold still, most of them are inapplicable, and others border on the barbarous. The young child should be completely anæsthetized by *ether*, and then placed in the *sitting position* on the lap of an assistant, with its head against the left shoulder. The mouth is kept open by a gag similar to those furnished with sets of intubation instruments. Three or four pairs of forceps, either the author's (Fig. 13) or other modification of Löwenberg's instrument, being in readiness, the left index finger is passed behind the velum, followed by forceps held in the other hand; a portion of growth is located, grasped, and

FIG. 13.



Author's Forceps.

removed, when, without withdrawing the guiding finger, quickly a second, third, and even fourth pair of forceps are used, and thus several pieces extracted before active hæmorrhage ensues. Instantly, then, the patient is tilted well forward with the head pendent to permit the blood, while flowing actively, to escape by the nose and mouth. In a few seconds the gush is over, the patient can be raised, the remaining blood cotton-swabbed from the pharynx, and the procedure repeated, and still again repeated, until the naso-pharynx is completely cleared. As a final stage remaining shreds are thoroughly scraped by the finger-nail.

Little fear need be entertained of blood running down the trachea. That which trickles slowly will course along the œsophagus into the stomach, and at times of rapid flow this danger will be obviated by the method of tilting the child well forward to permit of escape through the nose. Otherwise the blood is liable to gush into the trachea rather than to be swallowed, assertions to the contrary notwithstanding; for the function of deglutition during profound anæsthesia is suspended. Rapid and persistent cotton-swabbing may suffice, but is not so completely effective, and it prevents the reapplication of the anæsthetic during the bleeding interval, so prolonging the operation. The patient should be kept in bed until the following day, and during healing the parts should be cleansed by syringing through the anterior nares with an antiseptic alkaline solution.

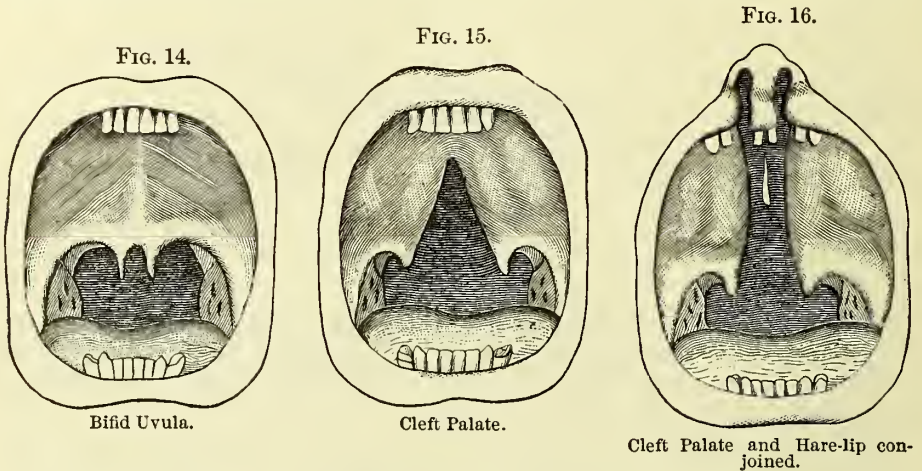
When the adenoids are small and soft, sufficient palliation perhaps, but not an absolute cure, can be effected by thorough and rapid scraping with the cleansed finger-nail, used as a curette, without the administration of ether. Gottstein's knife and Hartmann's curette, when deftly plied, can also be made effective without anæsthesia, but are apt to terrorize both the child and its parents.

Syrup of iodide of iron, internally, tends to correct the underlying dyscrasia—lymphatism.

IX. CLEFT PALATE.

True cleft palate is a congenital fissure in the roof of the mouth, of variable extent. The so-called acquired cases differ therefrom in presenting an unequal, ragged, or incomplete cleft, such as would be produced by the destructive ulcerations of syphilis. The extent of congenital cleft may vary from the slightest manifestation, that of a bifid uvula, to the grossest form of conjoined cleft palate and hare-lip, in which the fissure involves not only the velum palati and hard palate, but penetrates one or both sides of the alveolar arch and upper lip, with the presence of a separate intermaxillary structure. This article,

however, will not embrace the subject of hare-lip except incidentally (Figs. 14, 15, 16).



Etiology and Pathology.—Nature fails to complete her design as originally intended, and the defect doubtless dates from an early period of intra-uterine life. It is assumed that the same causes which produce rickets in children are prone to effect cleft palate. A deficient supply of phosphates in the diet of the mother, or failure on her part to thoroughly assimilate the phosphatic elements, may be regarded as an exciting cause.

Vander Veer states that “several years ago the lions in the Zoological Gardens of London were fed upon flesh containing too large bones for them to break and swallow, as is their custom. The young born while this method of feeding was pursued were observed to have cleft palates, and lived but a short time. The lions were then fed upon small animals, whose bones they could break easily, and the young born afterward had perfectly-formed palates.”

Intermarriage and unfortunate “maternal impressions” are also stated to be exciting causes. Whatever may have been the causes of the original inception of the malformation in previous generations, there can be no doubt that heredity now serves as a potent predisposing cause. In my own cases I have nearly always been able to elicit histories of other cases in other branches of the family. Vander Veer, Lawson Tait, and Gurdon Buck emphasize this fact. It will often reappear after skipping one or more generations, or it will diverge into collateral branches.

Symptoms.—The symptoms consist of an inability to nurse or to swallow perfectly, and, later, to talk properly—disabilities, of course, which vary in accordance with the extent of the cleft. A peculiar nasal intonation of the voice is occasioned, which, if the cleft be an extended one, will first attract attention to the defect in the crying of the infant, and later in life will characterize the speech. In swallowing, fluids regurgitate through the nose.

On inspection in marked cases the parts appear as if there were no soft palate, the side flaps being retracted by muscular tension, leaving a wide, inverted V-shaped opening, through which are visible the posterior and superior walls of the naso-pharynx with their covering of adenoid glandular tissue.

Treatment.—This may be considered in three divisions: prophylaxis, palliative measures, and operative treatment.

Prophylaxis.—Whenever any hereditary tendency to cleft palate, however remote, can be established, it would be a rational precaution to provide in abundance for the mother those articles of diet which are rich in phosphates—*e. g.* oatmeal—and to administer precipitated phosphate of calcium in powder, five to ten grains twice daily. It should be given, however, without the knowledge of the mother concerning the end in view, in order not to excite in her a “mental impression” toward cleft palate. For the same reason, in order to avoid directing the mother’s thoughts into this channel, she should not personally be questioned relative to heredity, or the subject be given prominence in any way in conversation with her during the period of gestation.

Palliative Measures.—If the cleft be large, some provision will be necessary to facilitate nursing. A large rubber nipple or one which is large and flat, so as to serve at the same time, while nursing, as a temporary obturator to close the cleft and permit of suction, is generally the best device. Such a nipple can be attached to the glass shield of an ordinary artificial nipple, commonly used to protect the mother’s nipple when nursing is painful, thus enabling the child to nurse indirectly from the breast, or it can be used with a nursing-bottle. In this latter case the bottle can be supplied with mother’s milk, at least for a time, by the preliminary use of a breast-pump. In extreme cases, especially those which are conjoined with the worst forms of hare-lip, it becomes necessary to feed the child by a spoon or feeding-cup, which is a laborious undertaking, but one likely to result successfully if it be properly carried out. Vander Veer mentions two cases, “son and daughter in one family, where the mother, for nearly two years in each instance, was obliged to give nearly her entire time to their care as regards feeding before they could help themselves.”

Later in life, if for any reason the operative treatment be not adopted or if operations should fail, much may be done to lessen the disability by the skilful adaptation of an obturator—a dental plate so constructed as to cover as much as possible of the cleft. A skilful dentist will fashion one to fit accurately and to extend quite far posteriorly, made of firm material, such as hard rubber or gold, furnishing thus a substitute for the hard palate and to a slight degree for the velum palati. But an obturator at best is but a poor substitute for a natural palate; it mitigates, but does not remedy, the defect; and to adopt permanently the use of one in lieu of a radical surgical operation is but to condemn the patient for life to the employment of a more or less troublesome and incomplete appliance.

A radical surgical operation, if it be skilfully managed, will be ultimately successful in a large majority of cases, and its dangers are slight in comparison with the disadvantage of a perpetuation of cleft palate for a lifetime.

Operative Treatment.—On account of the difficulty in phonation the operation for closure of the cleft should always, when possible, be performed early, before the child has learned to talk in an imperfect manner; otherwise, even though the cleft be closed later, much difficulty is experienced in teaching correct articulation. It should therefore be performed between the ages of one and a half and three years.

The operation is known as staphylorraphy when the cleft involves the soft palate only or extends but little into the hard palate; and osteoplasty when the palate process of the superior maxilla is so deficient as to necessitate the Ferguson procedure of drilling off edges of bone to bring together in the centre.

It is not my purpose to speak of this operation in detail. It is one which has interested the greatest surgeons of the day, and which will be found described at length in all text-books of surgery. But there are certain points

essential to obtain a good result—that is, perfect primary union of the two sides—and these salient features of the operation will be described.

It is important that the general condition of the patient be good, and that the season of the year be favorable; that is, preferably, not during the heated term of summer. The bowels should be opened freely the day before the operation by the administration of castor oil the night preceding this. Special care should be taken to avoid vomiting, caused by the anæsthetic, by forbidding any breakfast on the morning of the operation. One can readily understand that the whole success of this long and tedious operation will depend upon securing primary union, and that this preliminary treatment is calculated to insure a condition of health favorable to such union.

For anæsthesia in operations about the mouth chloroform is often preferred to ether, because its administration can be more interrupted; but children with cleft palate are apt to be generally feeble, so one must consider ether the safer anæsthetic for prolonged use; but one can commence with chloroform, because of its greater rapidity and pleasantness of action, and continue, as soon as unconsciousness is secured, with ether.

As with most other operations on the mouth and throat, the patient should be placed in Rose's position; that is, with the head pendent from the edge of the table, and the shoulders elevated by a small hard pillow, so that blood will gravitate into the naso-pharynx and not into the windpipe. In this position, at times when hæmorrhage is freest, the patient can be rolled upon the abdomen and the blood allowed to flow from the mouth and the nose.

The most suitable gag is Mussey's modification of the Whitehead gag. It has a tongue-depressor attached,—a matter of importance as it is absolutely necessary that the tongue be held depressed at the same time that the mouth is gagged open. The tongue-depressor of the Whitehead gag is attached by a hinge-and-ratchet joint, which easily gets out of order, and detracts from the value of the mechanism. In the Mussey gag the tongue-depressor is a part of the same piece, but by force it can be bent to a different angle if required.

Many and complicated needles have been devised for the purpose, among which may be mentioned Prince's needle as ingenious, but rather complicated. All that is necessary is a curved needle mounted in a handle, as illustrated in Fig. 17. This needle is often kept in the shops, but the curve should be much

FIG. 17.



Author's Modified Staphylorrhaphy Needle (half size).

more acute than is usually supplied, and the eye of the needle as near as possible to its point. These may seem like small details, but the selection of the needle is one of the most important points to insure a successful operation, inasmuch as it is sufficiently difficult to place the sutures with a perfect needle, and with a faulty one it may be impossible.

The patient being thoroughly under the influence of the anæsthetic, the first, and absolutely necessary, step is the division of certain muscles. This should be the first step of the operation, and not the last, for the reason that firm and accurate coaptation of the edges can be made only after the perfect relaxation of the muscles thereby produced. Having introduced the gag, one will notice the wide aperture in the roof of the mouth, and that it is seemingly impossible to draw together the two sides of the palate. This is by reason of the constant contraction of the palatal muscles. If one were to draw the two

sides together forcibly by means of stitches under the partial relaxation produced by the anæsthetic, they would only be ripped out again at the first effort of the child in crying or coughing or swallowing. Only perfect relaxation of the velum can assure complete primary union of the parts. The muscles to be divided on each side are the tensor palati, the levator palati, the palatoglossus, and one of the palato-pharyngei. The last-named muscles constitute the anterior and posterior pillar of the fauces respectively. The tensor palati arises on each side at the base of the internal pterygoid process, and, descending, its tendon winds around the hamular process, which can be felt by the tongue just to the inner side of the upper third molar tooth, and then spreads through the body of the velum. The levator palati has its fibres distributed just behind the tensor. A puncture, therefore, through the velum, commencing at the point of the hamular process, and following its curve about three millimetres upward, will sever the tendon of the tensor. Then the knife, with its cutting edge directed upward, should have its handle depressed, withdrawn, reintroduced (in the same opening), the cutting edge directed downward and handle elevated, cutting, in this manner, the posterior surface of the velum more widely than the anterior surface, and so severing as many fibres as possible of the levator. Having done this, one will notice how much more easily the two sides can be approximated.

Next raise the velum on each side and snip with scissors the anterior pillar, and, *on one side only*, the posterior pillar, in order to guard against atrophy of the palate by leaving the arterial supply intact on the opposite side.

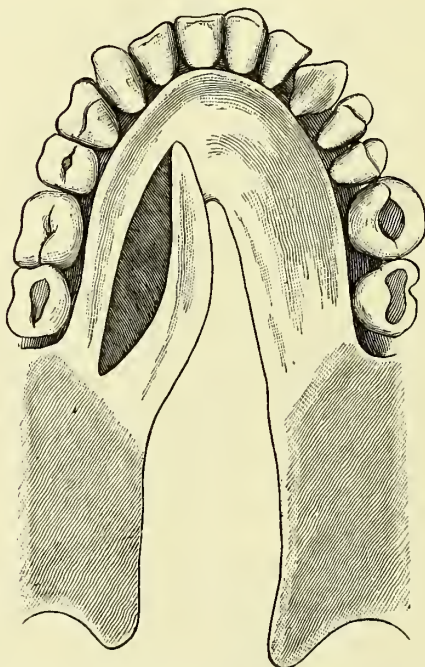
The hæmorrhage which is caused by these punctures is not likely to be dangerously profuse, although a small artery is severed, but the galvano-cautery point-electrode subsequently introduced would serve to arrest an excessive bleeding.

The edges of the cleft should be thoroughly pared, as merely splitting them does not result in the same satisfactory raw surface, and then provision can be made for closing the cleft in the hard palate. If this part of the fissure be not very extensive, the Warner-Langenbeck method is best. Loosen tissue to slide to the median line by making lateral incisions through the mucous membrane and periosteum, and by incision around and behind the anterior end of the cleft, extending to the bone both on the buccal and nasal surface; then, by means of a periosteal elevator raising the periosteum from the bone from the lateral incisions to the edges of the cleft, which part of the edge must also be well detached from the underlying bone and fascia, and properly freshened wherever it is to join the one of the opposite side (Fig. 18). These two flaps can then be brought together in the median line.

For extensive bony clefts Fergusson advocated the separation, by boring and chiselling, of sufficient of the bony edges to bring together in the centre to close the cleft. This procedure appears unnecessarily formidable, apart from the fact that with very wide cleft—the cases with which the ordinary flap operation is inadequate—the bony edges are likewise too scanty to afford a reasonable prospect of success. For such wide clefts the soft flap method recently proposed by Davies-Colley, of Guy's Hospital, commends itself. Figs. 19 and 20 will convey his idea.

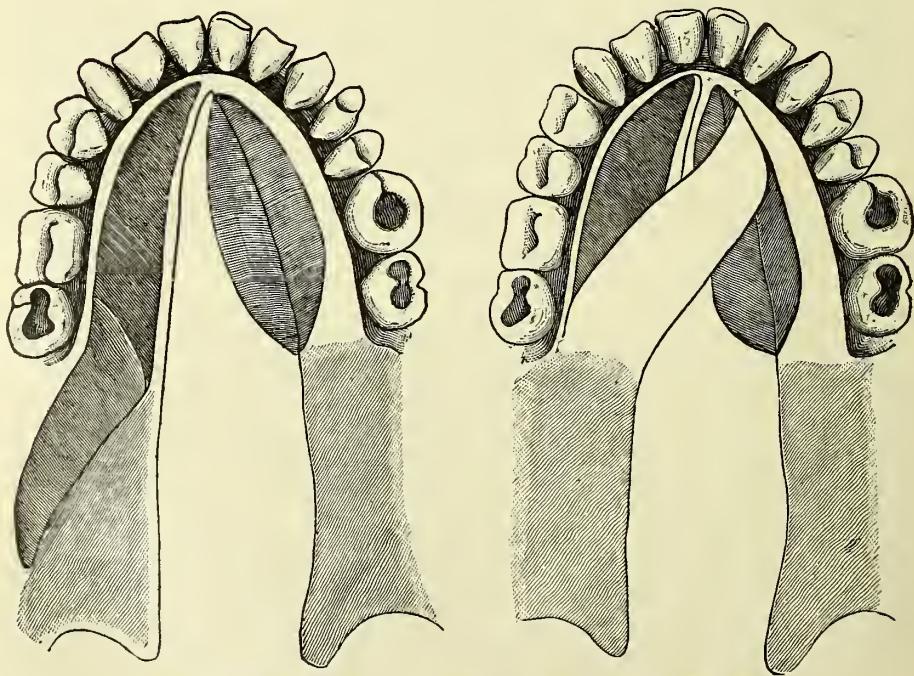
Next, the introduction of the sutures, by far the most difficult part of the operation. I prefer silk sutures, and consider them much superior to silver wire and shot, as they are softer in the mouth, and seemingly do not produce the same amount of irritation and annoyance to the child. Two colors, white and black, should be used, as all the stitches should be passed before tying, and if these colors alternate confusion of the ends need not occur. The well-curved

FIG. 18.



Warner-Langenbeck Method of Closing Small Bony Clefts, flap prepared on one side only.

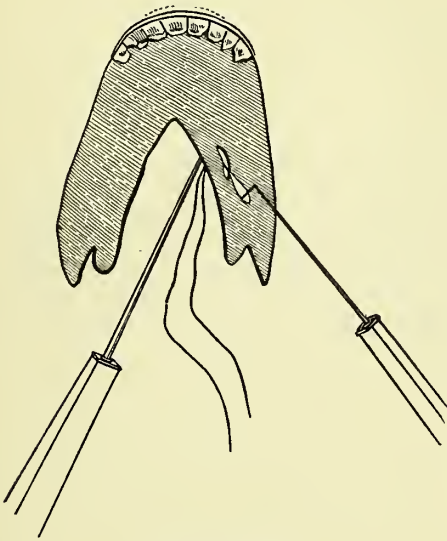
FIGS. 19 and 20.



Method of Closing a Wide Cleft of the Hard Palate (after Davies-Colley).

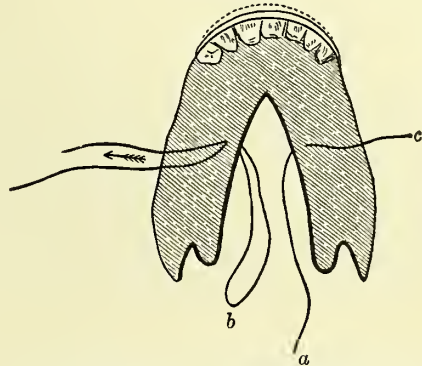
needle, having been threaded, is introduced on one side (the patient being recumbent), from below upward, or what would be, if the patient were upright, from behind forward (Fig. 21). To facilitate passing the needle the flap is held and drawn tense by forceps. The thread is then caught from the eye of the needle by a blunt tenaculum (Fig. 21), one end drawn all the way through, and the needle passed back and drawn off the other end.

FIG. 21.



Passing a Suture.

FIG. 22.



End of suture, *a*, is next passed through loop, *b*, which is used only to draw end *a* through the flap of that side. Ends *a* and *c* are subsequently tied.

This procedure is easier than if the needle were previously passed in the reverse direction, as is usually recommended. Having passed the suture on one side, one must pass a double thread on the opposite side, drawing up in like manner with a tenaculum the two free ends, which leaves the loop below (Fig. 22); the needle is then drawn back as before and disengaged. Then through the loop is passed the lower end of the single suture, and, by means of the double thread, it is pulled through the opposite side. In passing the stitches great care should be taken to engage sufficient tissue, not getting them too near the edge, and also to have them passed as nearly as possible at points opposite each other.

Before tying the sutures special care should be observed to see that the edges of the flaps are clean and free from clotted blood. Then, commencing anteriorly, the sutures are tied first by means of a slip-knot pushed down by the finger, the suture well tightened, and again tied by an ordinary knot. As the sutures, one after another, are thus tied, see that the edges are not turned in so as to bring mucous-membrane surfaces together instead of freshened edges.

Failure to unite by primary union is probably due to incomplete division of the muscles more frequently than to any other one cause; but the good health of the child, the careful paring of the edges, and placing of the sutures are also essential points. If, however, complete union should not result at the first operation, we certainly should operate a second or a third time.

It is rare indeed, with ordinary care and skill, that partial union will not be produced at the first trial, and this will encourage both parents and surgeon to persevere to a complete result.

Concerning now the subsequent treatment of the patient: At the completion of the operation, before the patient has revived from the anæsthetic, a hypodermic of morphine should be administered. This to prevent, as far as possible, vomiting and excessive crying—in other words, to maintain quietude of the parts. I consider it best, although all authorities will not be in accord with this opinion, to keep the patient partially under the influence of morphine during the first three days, for the same reason. The stitches may be removed from the sixth to the tenth day. Some of them by the sixth day will have ulcerated out on one or both sides, but this matters not when primary union is secured; and if primary union is not secured, the stitches will not hold the parts together after the third or fourth day. But as a matter of precaution, to give some strength to the newly-formed union, the stitches may be left until the time stated. To facilitate their removal an anæsthetic should be administered.

[NOTE.—Since going to press the author has observed an instructive case of retro-pharyngeal lymphadenitis in an infant four months of age. The child was convalescing from infectious pseudo-membranous tonsillitis (folliculous) when dyspnœa commenced, and increased for two weeks, when suffocation was imminent. Voice was unimpaired and inspection of the fauces negative, but palpation disclosed a hard tumor projecting from the posterior pharyngeal wall in the median line, low down and pressing upon the opening of the larynx. Three punctures into this tumor failed to evacuate pus. Tracheotomy was immediately performed. Resolution was complete; at the end of two weeks the tube was withdrawn, and the child recovered.]

GASTRIC CATARRH AND GASTRIC ULCER.

BY A. D. BLACKADER, M. D.,

MONTREAL.

I. ACUTE GASTRIC CATARRH.

ACUTE GASTRIC CATARRH, otherwise known as acute gastritis, gastro-adenitis, acute dyspepsia, or gastric fever, is an acute inflammation of the glandular tissue of the stomach interfering with its digestive functions, and generally due to the presence of irritating ingesta. The attack is attended with pain, anorexia, and nausea or vomiting; frequently also by general pyrexia. It is occasionally complicated by reflex nervous symptoms of a more or less serious character. Associated disorder in other portions of the alimentary canal may be met with. While occurring at any age, artificially reared infants and delicate children are especially prone to this disorder.

Etiology.—During infancy the stomach appears to be peculiarly liable to disturbance of its functions. It is the period of its most rapid development, and not only does it increase in size, but it has to assume more varied duties. At the same time, the demands upon it, incident to the very rapid growth of the body at this period of life, are proportionately larger than at a more advanced age. Infants fed at the breast generally escape, but not always. Occasionally errors in diet on the part of the mother, violent disturbance of the nervous system, or the appearance of the catamenia, may produce such changes in maternal milk as to render it less digestible, and thus bring about an attack of acute catarrh in the infant. It is, however, among those who have been artificially fed from the early days of infancy that disturbances of this character most frequently occur. The essentials of artificial feeding in infancy,—a milk, practically sterile, containing the proper amount of albuminoids, fats, and sugars, fed to the infant in proper amounts, at a proper temperature, and at due intervals, so as to permit perfect digestion with a short period of rest for the stomach,—have not yet been generally attained, even in our more intelligent families; while, among the poorer classes, how often does the infant's food fail in every one of these details! During infancy, also, appear the reflex nerve-disturbances generally attributed to dentition. Certainly at this period acute disturbances of the stomach are more frequently met with than either before or after.

By the end of two years the powers of the stomach are more developed; the demands of the system less exorbitant; any irritation accompanying dentition is past; and, under a careful dietary, attacks of acute catarrh should be infrequent. The rich and varied table diet often injudiciously allowed after this age may, however, conduce to an attack.

Generalizing, we may say that any excess in the amount of food, too great variety in its character, the use of such stimulating food as highly-spiced dishes, pickles, or sauces, irregularity in the meal hours, or the unregulated and unlimited eating of fruits, cakes, or sweetmeats, especially between meals, may in

children bring on an attack of acute indigestion. Food or drink, too hot or too cold, quickly taken, may also occasionally be an exciting cause.

Closely associated with errors in the dietary as an etiological factor is the imperfect mastication so often given to food. Children require to be taught to masticate, and their teeth from the time of their first appearance should claim the careful attention of the attendants.

There is not, however, in all children an equal susceptibility to disturbance. Some appear to have particularly vigorous stomachs which tolerate much abuse, while it is only with the greatest care that attacks can be averted in others. In some a predisposition to weak digestion is distinctly hereditary. Anæmic children are peculiarly prone to attacks. The close association between rickets and disorders of digestion has long been recognized. The scrofulous and rheumatic diatheses are also predisposing factors. Unsanitary conditions of life markedly impair the digestive powers, and thus favor an acute disturbance; especially is this true of want of exercise in the open air.

The acute ailments and specific fevers of childhood frequently leave the mucous membrane of the alimentary canal in a weakened condition, from which it takes time and a very careful dietary to thoroughly recover. Of this class of disease Ewald says: "Although the gastric symptoms are relegated to the background by other manifestations, yet in those cases with dyspeptic disturbances, in which we are enabled to examine the organ soon after death, we will find the anatomical changes of acute gastritis."

In some children the sudden checking of the cutaneous circulation, by chill from imprudent exposure, may occasionally interfere with the process of digestion and bring about an attack. Eustace Smith thinks this a very frequent cause of trouble. In our more severe climate children are more perfectly clothed in flannel than in England, but in children with weak stomachs I have frequently noticed an attack of gastric catarrh brought on by getting the feet damp. Unless due care be exercised, one attack may predispose to others.

Pathology.—Our knowledge of the minute changes in the mucous membrane in acute gastric catarrh has, until lately, been very limited; so much so, that some writers have questioned the propriety of admitting this among the list of actual diseases. In his recent work Ewald protests against the use of the word "catarrh" as creating an erroneous conception. "The structure," he says, "of the gastric mucosa, better designated the glandular layer, *tunica glandularis*, is such that it is out of the question to call it a mucous membrane in the ordinary meaning of the term. . . . It is simply a peculiar feature of the inner layer that the protoplasm of the epithelium of the excretory ducts possesses in a remarkable degree the property of being converted into mucus. . . . Dr. Beaumont's investigations on his patient, St. Martin, showed that every catarrh, even the mildest, was accompanied by a disturbance of the secretion of gastric juice; consequently by an affection of the glands themselves. The inflammation is thus not catarrhal, but parenchymatous and interstitial. It has nothing in common with a catarrh except the "flow," the secretion of a more or less abundant, but always alkaline, transudate into the cavity of the stomach. Misled by the term "catarrh," we are too prone to underestimate the importance of these processes, particularly when they are chronic, and by thinking, for example, of a chronic pharyngeal catarrh, we lose all proper standards of comparison."

Macroscopically, the mucous membrane in acute catarrh appears swollen and reddened. In severe cases slight hæmorrhages, or even small erosions, may occur; the submucosa may be œdematous. Microscopically, there appears an infiltration of the interstitial tissue with leucocytes; the differentiation between

the parietal and the principal cells can no longer be made out, while all the cells may alike be seen to have become granular and cloudy, and in part separated from the *membrana propria* of the glands. The mucous cells are especially abundant in the pyloric region, and extend down deeply into the ducts of the glands.

Symptoms.—Cases of acute gastric catarrh have been divided into two classes, the *febrile* and the *afebrile*, according as they are, or are not, accompanied by pyrexia. The division is a convenient one. The febrile are much the more severe. The afebrile run a short, mild course, and are as a rule unaccompanied by serious symptoms.

The onset of an attack is generally sudden. Within an hour or two after the error in diet the child shows signs of being unwell. If an infant, after a short sleep it awakes crying and apparently in pain. Its thighs are flexed on the abdomen. It moves restlessly from side to side, and whines piteously or cries bitterly. The temperature will be found more or less elevated, 102° to 104° , the pulse and respiration quickened, the tongue furred, the abdomen distended, and pressure on it evidently increases the child's distress. The bowels at this time may, or may not, show signs of disturbed action. Vomiting generally occurs early, with some temporary relief. After this the infant, if allowed, may eagerly take the breast or its food again, only to reject it, curdled and sour-smelling, after a short interval. If the ejecta be carefully examined, there will be found a marked deficiency of hydrochloric acid, and in its place the presence of lactic and butyric acids. Vomiting may recur several times; at the last, watery, sour-smelling mucus, perhaps more or less bile-stained, being ejected. There is now complete anorexia. The infant is restless and feverish, if not actually crying with pain, and its sleep is much broken and disturbed. Under proper treatment the attack is generally of short duration, and in twenty-four or forty-eight hours a few loose movements carry away any of the offending material that has escaped into the bowel; the fever subsides; the infant again sleeps quietly; but for a few days it is less eager for its food, which it is inclined to take more slowly and in smaller quantity.

In older children the attack manifests itself by a feeling of listlessness, with more or less drowsiness. The child will give up its play and prefer to lie down. Uneasy pain in the epigastrium is soon complained of, with a feeling of nausea and headache. If the child fall asleep, it is a very disturbed sleep, from which it frequently awakes in a fright, complaining of bad dreams. Dark circles may now be noticed under the eyes; the face is generally pallid unless the fever runs high. In that case a peculiar pallor about the upper lip and the *alæ nasi* is very distinctive of irritation of the stomach. The tongue is coated heavily toward the base, but the tip and the edges are red; the skin is dry; the pulse is quickened; the temperature may be high— 103° to 104° —but if so it reaches its height early; the abdomen is distended, pressure over the epigastrium increasing the uneasiness; and the breath is generally heavy or sour-smelling. The secretion of saliva is increased, so that during sleep it may dribble on the pillow. Vomiting may occur, but not so generally as in infants. When it does, there is usually much retching, and toward the close biliary matters, with watery mucus, are ejected with much straining. The bowels are constipated and the urine scanty and high-colored, with an abundant sediment of lithates. The headache is generally frontal, although sometimes temporal. In some cases an associated pharyngitis may be noticed; in others a few herpetic vesicles appear on the lips. In mild cases the attack subsides in a day or two, but in the more severe forms the fever may persist for four or five days, leaving the child in an exhausted state, from which, however, under careful

dietary, it generally recovers rapidly. Occasionally an attack of acute gastric catarrh is followed in a few days by catarrhal jaundice. The inflammation has probably extended down the duodenum, blocking the common bile-duct. Such cases are usually of short duration.

Although, in general, an attack of acute gastric catarrh may give us little anxiety, at times we have associated reflex symptoms of a very alarming character. The convulsive seizures of infancy, dependent so frequently upon gastric or intestinal irritation, are familiar to all and require prompt treatment. The danger of cerebral hæmorrhage during such an attack should always be borne in mind.

In older children more alarming, because more unusual, symptoms of reflex irritation are occasionally met with. In some instances localized or diffuse clonic muscular movements have their origin in gastric irritation. Symptoms closely resembling those of meningitis have been reported by Seibert. Fraenkel relates the case of a child four years old who shortly after eating a large amount of table food lost the power of movement and sensation on the right side. Complete recovery followed on the next day. Henoch records a case of complete aphasia in a child which passed away an hour later after the vomiting of some undigested fruit. Such cases, however, are rare, and should always receive the most careful attention on the part of the physician, lest, instead of being reflex, they arise from a distinct and all-important lesion.

Diagnosis.—In most instances, with a distinct history of some error in diet, no serious difficulty will be experienced in arriving at a guarded conclusion. The sudden onset, the tenderness over the epigastrium, the relief afforded by vomiting, and the rapid subsidence of the symptoms will in a day or two enable us to assure the parents that no more serious trouble need be apprehended. In cases attended with fever, however, it is always wise to speak more or less guardedly at the first. The onset of scarlet fever should always be excluded. In this disease we have as an early symptom a definite amount of congestion of the fauces, followed frequently by some enlargement of the glands at the angle of the jaw. The irregular erythema, sometimes appearing for a few hours in disorders of the stomach, should be distinguished from the scarlatinal eruption with its more regular development and longer duration. In doubtful cases, for such will arise, isolation for twenty-four or forty-eight hours will solve the problem. Tonsillitis and diphtheria may, with care, be easily excluded. An attack of acute catarrh may closely resemble the onset of pneumonia. J. Lewis Smith relates a case in which the high temperature and expiratory moan simulated a pulmonary inflammation, but was promptly relieved by the expulsion of some orange-pulp. In cases such as these careful attention must be paid to the pulse, the respiration, and the temperature. In typhoid fever the rise is more gradual; we frequently meet with an initial bronchitis, the prodromata are more marked, and some enlargement of the spleen may be made out. In acute gastric catarrh the onset is more sudden, and the distention of the abdomen more marked, than is general in typhoid fever at an early stage, while tenderness is noted in the epigastrium, not in the iliac region, and the temperature falls after a few days. In delicate children the possibility of tuberculosis must always give us anxiety. We have no absolute symptoms by which we can exclude this disease. A slow pulse may occasionally be met with in gastric disorder from irritation of the vagus. The vomiting of meningitis is, in general, indistinguishable in its character from the vomiting of mere gastric irritation, and the condition of the tongue is no certain guide. Under these circumstances a careful watch for localizing symptoms will be required, and a very guarded opinion must be given.

Prognosis.—The prognosis of acute gastric catarrh must be regarded as very favorable. Only in delicate infants, whose hold on life is extremely frail, will the disturbance of nutrition or the gastric irritation threaten immediate serious results. Such an attack may be the beginning, however, of a gastro-enteritis, which may prove fatal. Convulsive seizures are always serious. Relapses are common in artificially-fed infants and in older children unless due care be exercised.

Treatment.—In acute gastric catarrh the first important indication for treatment would appear to be the removal of the offending material in the stomach. Nature in many cases effects this spontaneously by the induction of vomiting. Should we see the case early, before vomiting has taken place, we may favor it by the administration of ipecacuanha, either in the form of a powder, or of the wine or syrup. If some hours have elapsed, however, a large portion of the offending stomach-contents may have escaped through the pylorus, and a gentle but prompt purgative is then called for. The following are suitable prescriptions under the circumstances:

R̄. Hydrarg. chloridi mitis gr. ij-iv.
Sodii bicarbonatis gr. xij.—M.
In pulv. iv. divid.

Sig. One every three hours until a free evacuation of the bowels is secured (for a child of three years).

Or, R̄. Hydrarg. cum creta gr. vj.
Sodii bicarbonatis gr. viij.
Pulv. rhei gr. viij.—M.
In pulv. ii. divid.

Sig. One to be given immediately (for a child of three years).

Or, R̄. Sodii et potassii tartratis gr. xxx.
Sodii bicarbonatis gr. iij.—M.
In pulv. vj. divid.

Sig. One to be given every hour in a wineglassful of hot water until a free evacuation is secured (for a child of three years).

In those cases where vomiting is troublesome and persistent minute doses of calomel, or of calomel and soda, may be given dry on the tongue. My own preference is for the triturate of a tenth of a grain, or of the tenth of a grain of calomel with a grain of soda, to be given hourly until eight or ten doses have been taken. This generally checks vomiting and secures a free evacuation of the bowels within twelve or twenty-four hours. It probably serves also to check to some extent the development of bacteria in the stomach. Should pain in the epigastrium be complained of, a warm poultice of linseed-meal, either pure or with a proportion of mustard, applied over this region, will be a source of much comfort. After the first acute symptoms have passed off, a mixture containing sodium bicarbonate, with a minute dose of *nux vomica*, will distinctly favor a return to healthy secretion:

R̄. Sodii bicarbonatis gr. xlviij.
Tr. nucis vomicæ ℥vj.
Aquæ carui, ad f̄z iij.—M.

Sig. One dessertspoonful to be given four times daily (for a child of three years).

As the case progresses and the inflammatory action subsides, the amount of the *nux vomica* may be increased.

The dietetic treatment is even more important than the medicinal. After the stomach is emptied it should have complete rest for some hours. Water in small quantities or small pieces of ice should be all that is allowed until the inflammatory action has sufficiently subsided to permit the secretion of gastric juice. Any attempt to give the child food before this will only increase the existing hyperæmia. In general, after about twelve hours of abstinence, milk diluted with either Vichy or lime-water, may be allowed in small quantities at a time. Should it disagree, a weak broth with barley- or rice-water may be tried. The recourse to solid food must be gradual. Starchy food, which is principally digested in the small intestine, may first be given, while stronger nitrogenous food is withheld for a few days longer.

Should nervous symptoms, such as sudden twitchings or startings, make their appearance, great quiet should be maintained in the sick-room, which should be moderately darkened. At the same time an enema, containing bromide of potassium or chloral, or both, in a little starch- or gum-water, may be given to relieve the nervous irritability, and, if possible, to ward off any convulsive seizure.

As long as the pulse is quickened or the temperature elevated the child should be kept in bed. Afterward over-fatigue should be avoided, as tending to a relapse.

When the gastric irritation has quite subsided, the tongue become clean, and the appetite has to some extent returned, the administration of some ferruginous tonic, with a daily drive in the open air, will generally prove of distinct value.

II. CHRONIC GASTRIC CATARRH.

This disease, also called chronic glandular gastritis, or chronic vomiting, is a chronic inflammatory condition of the glandular tissue of the stomach, giving rise to a diminution in both the quantity and the quality of the true glandular secretion (hydrochloric acid and pepsinogen), but attended with the secretion, sometimes in large quantities, of an alkaline mucus which possesses no digestive powers. As a result of this condition we meet with, in time, an enfeeblement of the muscular coat of the stomach leading to the undue retention of food. Chronic gastric catarrh is undoubtedly the condition most frequently encountered in the chronic digestive disorders of childhood. Only very seldom at this period of life can such disorders be referred to a distinct neurosis.

Etiology.—The causes leading to the condition of chronic gastric catarrh in childhood are closely allied to those already mentioned as inducing an acute catarrh. The continued irritation of the gastric mucous membrane by the ingestion of large, imperfectly masticated and insalivated morsels of food; by the habitual use of food, indigestible or improperly cooked, such as hot bread or cakes, pastry, and fried dishes; or by the habit of eating sweetmeats at all hours of the day, may occasion this condition, either directly or indirectly, by producing acid fermentation in the contents of the stomach. Another source of irritation is the continued contamination of the food by offensive discharges from ulcerations in the nose, throat, or mouth; from decaying teeth; and from the muco-purulent discharges, often very considerable in amount, from adenoid growths. Repeated attacks of an acute or subacute form are very liable to lead to this condition, especially in children with lowered vitality living under imperfect sanitary conditions. The presence of anæmia, rachitis, or scrofula

may be regarded as distinctly predisposing; also prolonged convalescence from an acute inflammatory or specific fever.

Any engorgement of the gastric veins due to valvular heart disease or to chronic inflammatory disorder in the liver and lungs will, of course, distinctly predispose to this condition.

Pathology.—The conditions in chronic catarrhal gastritis are but an extension of those referred to under the heading of Acute Gastric Catarrh. The whole organ is usually enlarged. The mucous membrane, usually thickened, is of a pale-gray or slate-gray color, with insular deeply injected areas, and is covered with a closely adherent layer of mucus. In places, especially in the vicinity of the pylorus, the hypertrophied mucous membrane may form small papillary projections, the so-called *état mamelonné*. In more advanced stages this condition may give rise to distinct polypoid outgrowths. The minute anatomy, says Dr. Ewald, is that of a parenchymatous and interstitial inflammation, most noticeable in the pyloric region. The gland-cells may be found partly destroyed, partly granular, and partly shrivelled up. The differentiation between the principal and parietal cells is impossible. In many places the ducts have lost their regular form and show an atypical ramification. There is an abundant small-celled infiltration, most marked near the surface of the mucosa. The superficial layer of the epithelium of the mucosa is loosened, and can be separated in adherent shreds. The mucoid transformation of the cells of the tubules is a prominent feature, and may be observed to extend down to the base of the glands. Whether this degeneration may to any extent retrograde, or whether it is permanent, Dr. Ewald has not been able to decide.

As the disease advances changes in nutrition produce a progressive fatty degeneration of the cells, with finally complete atrophy of the mucous membrane. To this condition Dr. Ewald proposes to give the name of *anadenia of the stomach*. This atrophic process may advance in two ways: (1) by progressive destruction of the glandular parenchyma, so that finally nothing is left but a layer of small round cells, in which appear isolated remnants of the former parenchyma; (2) by a marked activity of the interstitial connective tissue, leading to hypertrophic proliferation, with much thickening of the walls, but with great contraction, so that the capacity of such a stomach becomes very limited.

In either form it is a severe irreparable process, which specially involves the glandular layer of the stomach, and which is characterized by a complete disappearance of the secretory parenchyma.

Symptoms.—The symptoms at first are those of impaired digestion. The appetite is lessened, except at occasional intervals, when it may appear increased. Ill-defined gastric distress and colicky pains, with distention of the abdomen, indicate the presence of fermentation. Nervous symptoms, such as headache, listlessness, irritable temper, and disturbed sleep, owing in great measure to reflex irritation, become manifest. General nutrition sooner or later begins to show signs of impairment. The child looks pallid, dark circles appear under the eyes, the muscular system is badly nourished, the pulse is wanting in tone, and slight exertion produces signs of fatigue. The indications of digestive disorder now become more prominent: the appetite fails at the regular meal-hours, but during the intervals there may be cravings for unsuitable food. The breath, especially in the early morning, is heavy-smelling; eructations occur during the day; pain, referred to the epigastrium, is frequently complained of; nausea, recognized by sudden pallor of the countenance, recurs occasionally, but vomiting in older children is infrequent. In

infants vomiting is often a most pronounced feature; hence the title, "chronic vomiting," often given to the disease. Constipation is generally pronounced, and is very difficult to relieve. The motions consist of hard rounded masses, of offensive smell and variable color, passed with much straining, and generally associated with an increased amount of mucus. Occasionally an evening rise in temperature may be observed, exciting suspicions of typhoid fever or tuberculosis. In the more severe cases, after the disorder has run a prolonged course, and the failure in general nutrition has become very marked, "the patient either literally pines away like a lamp the oil of which has not been replenished," or falls an easy victim to some intercurrent disease.

Such may be said to be a general picture of this disease. Its course, always very prolonged, is perhaps more irregular than that of most chronic affections. The stimulus of a season at the seaside, or in bracing mountain-air, may for a time make an improvement in such children, especially in the early stages of the disease, but unless we can secure the necessary watchfulness over the dietary and general hygiene, a fresh exacerbation is easily induced, with renewal of all the unfavorable symptoms.

In infancy symptoms of indigestion occasionally appear shortly after birth. Frequently the fault in such cases lies in the character of the nutriment supplied to the infant; but sometimes a feeble power of digestion appears to be inherited. Should disturbances of the digestive functions persist, the infant becomes restless, fretful, and colicky. Attacks of vomiting occur frequently; sometimes shortly after the food is taken, on some slight movement, the greater portion of the meal will be rejected, curdled and sour-smelling. At other times vomiting takes place some hours after the meal, and consists of watery mucus and lumps of hard curd or other undigested food. The appetite is variable; at times the breast or the bottle may be refused absolutely, and again food may be taken eagerly at first, but is shortly pushed aside with evident signs of distress. The face is pale, and, instead of the normal expression of placid content, it frequently puts on a pained look. The tongue is generally furred, but in infancy this is not so reliable a symptom as in older children. Sleep is fitful, much disturbed, and for short intervals only. Nutrition distinctly fails. Instead of the normal increase of from four to eight or ten ounces per week, the infant may scarcely hold its own or may even lose weight. The skin, along with the other tissues, suffers from lack of nutrition, the subcutaneous fat is absorbed, and the superficial veins show distinctly through its more delicate structure; the muscles are small and flabby; the extremities are with difficulty kept warm; the fontanelles, if open, will be found depressed; and the coronal suture may be prominent, owing to depression of the frontal bones. Constipation is a frequent symptom, but occasional attacks of diarrhoea occur, with the passage of undigested food and some mucus. Various forms of skin rashes frequently make their appearance, such as erythema, urticaria, and lichen. Vomiting in such infants, especially if there be much failure in general nutrition, is always a symptom requiring much attention. In my own experience it has almost invariably yielded to patient and careful treatment, but it quickly reduces the strength, and hydrocephaloid symptoms may supervene. Parasitic stomatitis is apt to prove a troublesome and, in a few cases, a serious, complication. If symptoms of indigestion persist and wasting becomes extreme, all our measures seem to fail, and the infant sinks into the condition known as simple atrophy.

A few of the symptoms met with in older children require more extended notice. Pain and uneasiness, referred to the epigastric region, is a very common complaint. In general, the distress becomes more pronounced shortly after taking food, but occasionally it seems to be more felt when the stomach is

empty. In all cases distinct tenderness is elicited by pressure on the epigastrium. The tongue is usually large and flabby, with heavy yellowish fur toward the base. The tip is red, and its papillæ reddened and prominent. Occasionally we meet with a comparatively clean tongue, or one marked by crescentic and wandering rashes. Too much importance must not be placed upon its appearance. The appetite is whimsical, variable, or sometimes altogether wanting. In some it is satisfied after a few mouthfuls, and afterwards requires much coaxing. Although a feeling of nausea is not an infrequent symptom, vomiting seldom takes place except on the occurrence of an acute exacerbation.

The sleep of such children is generally much disturbed. They toss about from side to side, dream, and talk in their sleep. Occasionally they may awake suddenly in great terror, and remain for some few minutes screaming wildly under the influence of fright, unable to recognize their attendants. Somnambulism in children is generally due to this same cause.

The amount of interference with nutrition that may take place is variable. Some appear to grow fairly well, though they remain pale; their frame is well developed, but the muscles are deficient in tone. In others nutrition is markedly defective. They are small for their age, their muscular tissue is poorly developed, and their pulse small, weak, and occasionally intermitting. Such children are liable to suffer from neuroses. Headaches, chiefly frontal, are frequently complained of; the temper is uncertain, and generally very irritable. Local muscular twitchings of choreic character are not infrequent. Syncopal attacks, closely resembling those of *petit mal*, are sometimes met with. Disturbances affecting the heart's action, or the respiration, have been reported.

The nutrition of the skin in such children is always impaired. Their skin is never clear, but is generally sallow in appearance.

A reflex irritation, referred to the nostrils or the anus, manifested by a constant picking of the nose or scratching at the seat, is very common and is extremely troublesome. A "stomach cough," generally due to an accompanying pharyngeal catarrh, is a not infrequent symptom. As a rule, it is most troublesome during the hours of sleep.

The course of chronic gastric catarrh in children varies much. After it has persisted for some time there is always a marked tendency to distention of the stomach, with impaired muscular action. This, if not checked, may go on to the production of actual dilatation, especially in those cases where the abdominal parietes are much relaxed. In some the large secretion of mucus becomes a prominent symptom, the disease extends to the lower portion of the alimentary canal, and general nutrition becomes still more rapidly lowered. (See article on "Mucous Disease.") In the severer forms of atrophy of the glandular tissue of the stomach the clinical picture may be that of a pernicious anæmia.

Diagnosis.—In the diagnosis of chronic gastric catarrh there should rarely be much difficulty. The long history, the epigastric tenderness, the disturbed digestion, and impaired nutrition, after the exclusion of organic trouble in the lungs, heart, or kidneys, will indicate clearly the character of the trouble with which we have to deal. If possible, however, an exact determination should be made by examination of the stomach-contents one hour after a test breakfast, which in older children should consist of bread and milk. The examination may be easily managed in infants by passing a soft rubber stomach-tube and withdrawing some of the contents. In older children this is more difficult, but may sometimes be managed. Advantage may be taken of any ejecta, or artificial means may be used to produce emesis. By this measure three forms

of chronic gastritis may be distinguished: 1. Simple gastritis, in which, after the test breakfast, hydrochloric acid is found in diminished quantity, while lactic and butyric acids are usually present. 2. Mucous gastritis, which differs from simple gastritis chiefly by the presence of a large amount of mucus. 3. Atrophic gastritis, in which the secretion of hydrochloric acid and pepsin is almost entirely absent.

In some cases of impaired digestion in infants it is necessary to exclude the presence of hereditary syphilis and tuberculosis. In the former possibility a decision should be arrived at without having recourse to medicines, as in simple gastric disorder a course of antisypilitic remedies may do harm (Pepper). In older children the presence of pyrexia, with the symptoms of chronic gastric catarrh, should always suggest the possibility of typhoid fever. The same considerations should influence us in forming a diagnosis in the case of chronic, as in that of acute gastric catarrh.

Popular opinion generally refers many of the symptoms of chronic gastritis to the presence of intestinal worms. In suitable cases it may be desirable to give a few doses of mild vermifuge to exclude their presence.

Prognosis.—The prognosis of chronic gastric catarrh in childhood, if placed under careful dietetic and hygienic treatment before the atrophic changes have proceeded too far, may be regarded as good. In infancy there is always danger of extension of the trouble to other portions of the alimentary canal. This is especially the case during the summer months. The continued interference with nutrition renders children more prone to the development of some intercurrent disease.

“While the dyspepsias of children are not of themselves often fatal, they are serious on account of the vulnerability of system they induce. They are prone to recur. They are apt to interfere with normal development, and to entail subsequent debility of digestion, of nerve, or of the entire nutrition” (Pepper).

Treatment.—The treatment of chronic gastric catarrh is in many instances one of the most unsatisfactory that we can undertake. The disease is apt to run a prolonged course and to have many relapses. The families in which we meet our more severe cases are frequently those who can only with much difficulty be impressed with the importance of strict attention to the details of treatment, and when we finally succeed in convincing the parents of the necessity of our rules, we find that the children refuse to be controlled.

Our first step in each case must be to investigate carefully all the factors, exciting and predisposing, tending to impair the functions of the stomach. The character of food taken by the child must receive our most careful attention, and, making due allowance for the idiosyncrasies of digestion so frequently met with among children, a systematically arranged dietary for the week should be drawn up, and rigidly adhered to, in each case. Instructions should be given that the attendant insist on proper mastication of the food. Nervous children especially are very apt to bolt it. Should the teeth be so defective as seriously to interfere with mastication, all food requiring it should be minced before giving it to the child. The amount also should be carefully regulated. I am convinced that many children accustomed to a richly-spread table have a tendency to overfeed themselves. Dinner, the heaviest meal of the day, should be taken about noon. The evening meal should be a light one. The general hygiene of the child will also demand the most careful attention if our efforts are to be successful. The child should live a quiet, regular life; it should retire early to bed, and its sleeping apartment should be cool and airy. The morning bath should be of a stimulating character.

For these children I prefer the bath recommended by Wiederhofer. The child on getting out of bed first receives a general rub down with a somewhat rough towel. It then steps into the bath, which contains warm water to the depth of three or four inches. It is afterward sponged down quickly with cool salt water, of which half a gallon or more is to be emptied over the chest and shoulders. When the sponging is finished, the child is then at once wrapped in a large towel and is briskly dried and dressed.

Children suffering from chronic gastric disorder are easily fatigued, and under the influence of excitement may readily over-tire themselves. This is to be avoided. At the same time, regular moderate exercise in the open air is to be insisted on.

There are several indications that should be considered in our administration of medicine:

1. The deficiency of gastric juice, which is generally met with in these children, may sometimes with advantage be supplied by the administration shortly after meals of hydrochloric acid with pepsin. In those cases where the tongue is coated with a white creamy fur an alkali, such as sodium bicarbonate, given shortly before the meal, appears to act as a sedative to the mucous membrane, while at the same time it stimulates to more active secretion the cells elaborating hydrochloric acid.

2. In almost all cases there is a deficient tone in the muscular coat of the stomach which calls for the administration of one of the vegetable bitters. My own preference is for nuxvomica, in smaller or larger doses as the case may require. Columbo, gentian, or quassia may also be employed, either in the form of infusion or tincture.

3. In many cases, owing to the large amount of mucus, fermentation either in the stomach or small bowel becomes a prominent feature, and the distention thus induced may, if allowed to persist, lead to a more or less parietic condition of their muscular walls. To relieve this aromatics may be added with advantage to our remedies, but some reliance may also be placed on antiseptics. Salol under these circumstances has, I think, given me very satisfactory results.

Should diarrhoea supervene, a combination of bismuth and salol will prove very serviceable. To relieve the colicky pains often complained of by these children some anodyne may occasionally have to be employed. I have also used with much benefit large enemata of warm water, as recommended by Ashby.

In infants, and sometimes in older children, vomiting becomes occasionally a troublesome feature, persisting in spite of treatment. Absolutely no food, under these circumstances, should be given by the mouth, all extraneous sources of irritation should be removed, and sedative enemata, containing small doses of either opium or bromide with chloral hydrate, may be given twice daily to subdue the nervous erethism. In these cases *lavage* of the stomach has sometimes proved a successful therapeutic measure. Dr. Booker, after a large experience in the Thomas-Wilson Sanitarium, says: "I believe stomach-washing is of undoubted advantage in the treatment of the digestive disorders of infancy. It has proved with me the quickest and most effective means for the relief of the vomiting, which I found generally relieved after the first washing; in only one case was it found necessary to stop milk food. The contra-indications to the use of the measure are heart disease and serious bronchitis or other pulmonary trouble. When the tube continues to excite vomiting and strong resistance, it is doubtful if advantage follows its use. A feeble condition of the infant does not necessarily contra-indicate the operation." In

older children lavage is rendered extremely difficult, owing to their determined resistance. Possibly results less efficient, but somewhat similar, may be obtained by the administration of warm alkaline drinks on an empty stomach. A combination of the potassio-tartrate of soda with a small amount of the bicarbonate, dissolved in hot water, may be given early every morning, or equal parts of Vichy and hot water may be taken once or twice daily. Sufficient time should be allowed for this to pass out of the stomach before food is taken.

In cases associated with constipation a determined effort should be made to secure a regular movement of the bowels once a day, with the least possible amount of irritation to the gastric mucous membrane. Some preparation of cascara may be given regularly at bed-time in doses sufficient to secure a daily motion of fair consistence. The action of the medicine should be favored by daily gentle massage of the large bowel, and by regularity in the time of soliciting a movement.

In children suffering from chronic gastric disorder any sudden chill of the surface should be prevented by the habitual use of a flannel binder over the abdomen. The extremities should be efficiently covered; the feet and ankles especially should be always dry and warm. Although ferruginous tonics, if symptoms of any acute exacerbation are present, may disagree, they may be given to many of these children with advantageous results.

III. GASTRIC ULCER.

Gastric ulcer is a lesion affecting the mucous membrane of the stomach, characterized by the formation of an ulcer of varying size and depth, and of uncertain position on the gastric wall. The disease in childhood may be indicated by symptoms similar to those met with in the adult—namely, epigastric tenderness, pain increased by the ingestion of food, and hæmatemesis. Occasionally the symptoms are very obscure, and a diagnosis is impossible until an autopsy reveals the cause of death. It is an exceedingly rare affection in childhood, and very few cases have been reported.

Etiology.—Gastric ulcer in children is generally associated with some constitutional disorder, such as tuberculosis, struma, and anæmia. Pneumonia and purpura hæmorrhagica are also mentioned as predisposing. Colgan reports a case in a child of two years and a half, due apparently to chronic gastric catarrh. Tuberculous ulcers are often multiple.

Symptoms.—According to Descroizelles, anorexia develops early and is steadily progressive. Vomiting may come on, but sometimes nausea only is complained of. Eructations and pain are frequently present, and the ingestion of food is generally followed by an exacerbation of the suffering. In some cases the symptoms are by no means distinctive. In one case reported the physical signs simulated those of pericarditis; in another, those of pneumothorax. In the case related by Colgan a well-nourished child had been in fair health up to the morning of the attack, when she complained of feeling unwell. Toward the evening she was seized with convulsions. When seen by Dr. Colgan her temperature was 106°; the pulse 150, rather full and tense; and the breathing stertorous. The convulsions were general, and there had been involuntary evacuations from both bladder and rectum. The convulsions were temporarily controlled, and consciousness, which had been lost from the beginning of the attack, was beginning to return, when a second attack occurred and terminated fatally. At the autopsy a perforating ulcer was found, with consequent peritonitis. The gastric mucous membrane was in a chronic catarrhal condition.

Gastric ulcer, dependent upon emboli from thrombosis in the umbilical vein, is said to be a frequent cause of hæmorrhage in the new-born.

Prognosis is very unfavorable.

Treatment.—The treatment, too, is generally unsatisfactory. If a diagnosis be made, the child should be confined to bed, and, if possible, it should for some days be fed only by the rectum with artificially digested food. Afterwards, a gradual return should be made to milk or bland starchy food, given in small quantities and frequently repeated. Of drugs, nitrate of silver in small repeated doses is probably one of the most satisfactory. Small doses of opium should be given to relieve pain. Gentle, soothing applications may be made over the epigastrium. If vomiting occur, bismuth is indicated.

GASTRO-MALACIA.

This term is applied to the softened, and sometimes ulcerated, condition of the stomach occasionally found after death in children. It is dependent upon the action of the gastric juice, which may happen to be present in the stomach at the time of death, upon the walls of the stomach itself, now dead and unprotected. Goodhart believes that an action may commence just prior to death, owing to a very defective circulation insufficiently protecting the tissues. Even if such be the case, it is the result of ebbing life, not a disease causing death, and as such it calls for no further remark.

MUCOUS DISEASE.

BY WILLIAM A. EDWARDS, M. D.,

SAN DIEGO.

THE fact that many different names and many etiological factors have been advanced to designate the train of symptoms and explain the pathology of the disease under consideration, serves to show that as yet there is not an entire consensus of opinion as to the proper classification of this condition.

Space forbids a complete recapitulation of the host of synonyms under which this disease appears in medical writings. We cite but a few: Chronic gastro-intestinal catarrh; Intestinal desquamative catarrh; Mucous disease; Chronic muco-colitis; Chronic croup of the intestines; Chronic follicular inflammation of the intestinal mucous membrane; Chronic pseudo-membranous gastro-enteritis; Mucous or Gelatinous diarrhœa; Mucous casts.

The term membranous enteritis has recently become somewhat restricted to a particular form of intestinal disorder characterized by irregularly recurring paroxysms of abdominal pain, unaccompanied by fever and relieved by the passage of membranous shreds or tubes, which for the most part are composed of mucin.

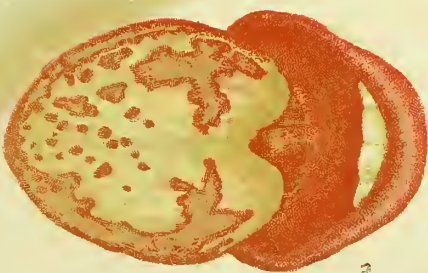
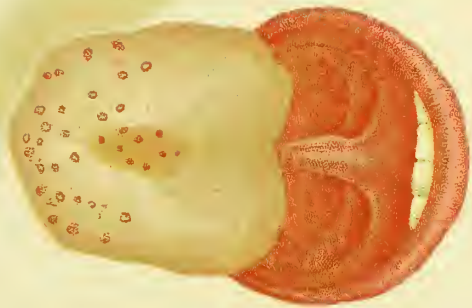
The present chapter will be restricted to a consideration of mucous disease as described by Eustace Smith of London, who defines it as an increased secretion of mucus from the whole internal surface of the alimentary canal: it is a mucous flux which interferes mechanically with digestion and absorption of the food, and by its influence in impeding general nutrition often excites suspicion of the existence of tubercle. This disease, unlike its analogue, membranous enteritis, is a very frequent condition among children, in whom it is most usually met with between the ages of three and twelve years.

Etiology.—The infectious diseases, particularly measles and scarlatina, but above all pertussis, may be followed by mucous disease—indeed Smith considers that pertussis, of all others, is the one to which this derangement can commonly be traced. It must not be forgotten that the mucous membrane of the alimentary canal in the child is naturally very active, and that the healthy stool in the young infant contains a large proportion of mucus; so that we can readily understand that if the child be habitually fed on indigestible food, thus presenting a constant source of intestinal irritation, the normal mucus may appear in abnormal amounts.

We must also remember that the stool of the healthy infant contains many bacteria and micrococci. Osler says that the most important are the bacterium *lactis aerogenes* and the bacterium *coli commune*.

In diarrhœal conditions the number is greatly increased. Booker has isolated forty varieties, and his conclusions are, that in the diarrhœa of infants not one specific kind, but many different kinds, of bacteria are concerned, and that their action is manifest more in the alteration of the food and intestinal

PLATE XII.



THE TONGUE IN MUCOUS DISEASE (LOUIS STARR).

contents and in the production of injurious products, than in a direct irritation upon the intestinal wall. So that from the authority of this careful observer, together with that of Jeffries and Baginsky, we may conclude that mucous disease is not bacterial in origin and does not arise from the presence of a specific micro-organism.

We, however, must state that Cornil considers that the peculiarities in all forms of membranous enteritis, mucous disease, and the like are not owing to different anatomical lesions, but to the difference in the nature of the micro-organisms: he considers that all forms are due to special micro-organisms introduced with the food.

Heredity does not merit consideration among the etiological factors, nor does climate, as the disease is seen in all countries and in all climes: it is perhaps more frequent in England than elsewhere, although this may be due to the fact that English observers have more carefully recorded their observations. It is usually seen in association with other diseases of the intestinal tract: an antecedent dyspepsia or a diarrhoea alternating with constipation is frequently noted before mucous disease becomes firmly established.

Day agrees with Meigs and Pepper that whether diarrhoea be caused by improper food, summer heat, dentition, or epidemic influences, the complaint, if it becomes chronic, is apt to terminate in mucous disease. It has been observed in children to follow typhoid fever, enteralgia, hæmorrhoids, and intestinal tuberculosis.

We ourselves have always accepted the statement of DaCosta that the disease was a manifestation of disordered nervous supply, which may be either general or local, and that the nerves presiding over nutrition and secretion are primarily at fault.

Wales is of the opinion that the primary seat of the disorder is in the ganglionic nerves of the intestines.

Certain it is that all of our cases presented marked evidences of deranged nervous action, and we agree with Goodhart, who considers the class of diseases that are the subject of this article under the title of "abdominal neuroses," and further states that he "is persuaded that although they may seem to be caused by temporary conditions—such as errors in diet—these varying pains and aches are often but the expression of a constitutional build. They are an evidence of nervous instability, and they are found in nervous children or in nervous families." In this observer's experience the children who are the subjects of mucous disease are the offspring of those whose nervous systems are feeble or diseased, or who are closely related to, or have themselves been, the subjects of fits, insanity, hysteria, neuralgia, rheumatism, or gout; or if not, have in themselves given other evidence of unstable nerves in the convulsions of infancy, passionateness, morbid timidity, chorea, or rheumatism.

Louis Starr, the American editor of Goodhart's book, favors the views of Eustace Smith.

Symptoms.—In the more chronic cases of mucous disease there is an almost constant sequence of symptoms. A coated, anæmic, flabby, and fissured tongue is usually observed, with aphthous ulcers of the mouth and tonsillar derangements.

Smith considers the appearance of the tongue to be absolutely characteristic: it appears to him as if brushed over with a solution of gum; this slimy look may be generally limited to a small spot in the centre of the dorsum. In my experience the whole tongue is more apt to be clean, stripped of epithelium, glazed or glossy. See accompanying Plate (XII).

All the stools do not contain mucus, but its passage may be paroxysmal, or

there may be an accumulation of mucus, and the discharges for several days may be made up almost entirely of this substance, or a great mass may be passed at a single stool; constipation may exist or this condition may alternate with diarrhœa. The patient may have only one mucous stool a day, or, as I have seen them, twelve to fifteen in twenty-four hours; after an attack of this kind the discharges are usually free from mucus for several days, or even for weeks, and the child apparently improves for a time, but only to suffer another exacerbation in a shorter or longer period. A simple enema or a mild aperient usually brings away large quantities of clear mucus or mucus stained by feces. These children rarely pass a normal fecal evacuation: the feces are apt to be soft, mushy, light-colored, and mixed with or coated over by mucus. The stools occasionally contain worms.

Some cases present certain premonitory symptoms before one of these large discharges of mucus occurs, as chilliness, blueness of the nails, tingling or pain at the finger-tips, dyspeptic symptoms, and a sense of uneasiness usually referred to the umbilical region. Smith has also observed that a most frequent seat of pain is over the left hypochondrium, and explains this by calling attention to the fact that at this point the colon makes a very abrupt turn, and the angle thus formed presents a site for the accumulation of flatus.

During an acute attack the former sense of uneasiness may become true pain; in some instances it is most severe: tenderness may extend over the entire abdomen, or it may be localized and developed only by firm pressure. Nausea or vomiting, in my experience, does not often occur, although it is mentioned by some writers.

The breath is usually most unpleasant, heavy and fetid; the tonsils are apt to be diseased, and no doubt contribute their share to this unpleasant odor.

The temperature is rarely above normal except perhaps at the height of a painful paroxysm; indeed an abnormal temperature would lead one to suspect some other and more serious condition, as phthisis. The surface temperature to the touch seems to be below normal, although the thermometer will probably not so record it.

The nervous system is early affected, and presents many evidences of derangement; hysteria in some of its many forms may exist, and night-terror with its peculiar concomitants, nocturnal incontinence of urine, somnambulism or the insomnia of gastro-intestinal origin, irregular muscular tremors, paresis, hysterical tetanus, neuralgia, hyperæsthesia, anæsthesia, convulsions, syncope, and stammering, have all been observed. Tinnitus aurium, transient defects in vision, as squinting, a disordered sense of taste, hæmorrhoids, prolapse of the rectum, and anal fissure have also been noted. The child's nature seems to have undergone a radical change: he is irritable and exacting; he suffers from mental depression, faulty memory, and hypochondriasis. In the older child melancholia may be noted.

Furuncles or carbuncles may arise, and sore mouth or herpes of the genitals are not unusual.

The appetite is at first increased, then becomes capricious, and finally almost complete anorexia exists; food produces distress by flatulent distention of the bowels, and it is only by the exercise of good tact that the little patient can be induced to eat at all. This, however, is not true of all cases: some children maintain their appetite throughout the disease, but, notwithstanding the enormous quantity of food consumed, the emaciation is extreme. The skin may have the characteristic hue of anæmia or the sallow tint of jaundice; it may be harsh, rough, and scaly. The urine is apt to be acid and to contain an excess of urates. I have not observed that the lymphatic glands in the neck are pecu-

liably liable to become enlarged on the slightest irritation, as stated by Smith, who also adds that they do not, however, necessarily suppurate or remain permanently swollen; the enlargement, after persisting for a variable time, may disappear completely.

It must be remembered that the little patient who is the subject of mucous disease does not present a regular sequence of symptoms, so that it is a difficult matter to present a didactic picture of the derangement; the symptoms are as erratic as the child itself. As Goodhart aptly remarks, such children are essentially angular in their moral nature and are an "odd lot." In this connection attention may be called to a paper by Ayres (*Med. News*, vol. lix., No. 1, 1891, p. 1) on chronic gastro-intestinal catarrh in relation to the etiology of some cases of insanity.

MICROSCOPIC APPEARANCE OF MATTERS PASSED.—They are very similar to the masses passed in cases of membranous enteritis, and are made up of opaque white solid masses, moulded or flattened, and small flocculent pieces of semi-translucent membrane. The tubes, branching membranes, casts, and fine network membranes are not seen in mucous disease. The description I have elsewhere given of membranous enteritis (in Keating's *Cyclopædia*, vol. iii. p. 166) also applies to the mucous masses voided in chronic gastro-intestinal catarrh.

Under a low-power objective the masses are seen to be due to the formation of mucous and epithelial matter (the cells having undergone fatty degeneration), and granular débris. H. B. Hare states that these matters are similar in chemical reaction to pharyngeal mucus, that they may possibly contain a trace of albumin, but no fibrin. Their surface may be seen to be composed of opaque and translucent parts; the former appear as rounded ridges marking off the latter into regularly arranged hexagonal or polygonal crypts.

Clark has observed that the product of diseased action on mucous membranes occurs in three varieties: first, clear, jellylike, and imperfectly membranous; second, yellowish, semi-opaque, flaky, and usually membranous; third, yellowish-white, dense, opaque, distinctly membranous, tough, and rather adherent to the subjacent surface.

Morbid Anatomy.—The morbid anatomy of the disease seems to be a thickening of the intestinal mucous membrane; there may be evidences of ulceration or enlargement of the glandular follicles of the colon or small intestine, the sigmoid flexure, and the descending colon, together with the lower part of the ileum.

Diagnosis.—As I have elsewhere remarked, if mistakes arise in the diagnosis of the affection, they are in all probability due to the carelessness of the observer rather than to any obscurity in the manifestations of the usual clinical phenonema of the disease.

The mucous masses may resemble and have been mistaken for *ascaris lumbricoides*; indeed, the parasite may be present in the discharges, as it finds in the mucus-loaded intestine a peculiarly acceptable habitat. The white, shining, detached pieces have been mistaken for segments of the various tape-worms, *tænia mediocanellata*, *tænia solium*, and *bothriocephalus latus*.

The lenteric discharges of dysentery have also been erroneously considered as illustrations of this disease; in scarlatina and tubercular disease mucous deposits are sometimes passed per anum.

The disease, however, of all others, with which we are apt to confound mucous disease is general or pulmonary tuberculosis: here it is that a carefully recorded series of temperature records is invaluable. In tuberculosis we find a continued elevation of temperature, while in mucous disease the temperature is

usually normal; at all events, it is only elevated during the height of a paroxysm, remaining high for two or three days and returning quickly to the normal.

Smith makes the statement that in some cases the temperature rises and remains elevated, perhaps permanently, although the symptoms in other respects correspond to those of mucous disease. I have never met such cases. He considers that these subjects are peculiarly prone to pneumonia, and that the deposit, only partially absorbed, undergoes cheesy transformation and forms the so-called pneumonic phthisis. Under these conditions I must confess that the differential diagnosis between mucous disease, pneumonic phthisis, and tuberculosis would indeed be a difficult problem to solve.

Prognosis.—Most cases run a prolonged and tedious course, with many recurrent attacks and exacerbations, extending sometimes into adult life. Absolute recovery rarely occurs.

Treatment.—The child that is the subject of mucous disease must submit to a constant supervision of its daily life. Its diet, regimen, and personal hygiene are of vital importance. The little patient must have a daily bath, first with castile soap and warm water, then a general sponging with alcohol, followed by an inunction of olive oil. In this way the peculiarly harsh, dry, and scaly skin can be restored to its normal function as one of the excreting organs of the body.

The diet always merits the most painstaking care; indeed, without a correct and suitable diet all other methods of treatment will inevitably fail. All sources of irritation are to be removed; easily-digested or even pre-digested food should be supplied, and the medical attendant should satisfy himself that undigested particles of food are not irritating the alimentary canal.

The following diet-table is taken from Eustace Smith (fifth edition, 1888), and is applicable to a child of seven years of age and upward:

Breakfast, 8 A. M. Three-quarters of a pint of fresh milk alkalized by twenty drops of the saccharated solution of lime; a thin slice of well-toasted bread; fresh butter; a fresh egg lightly boiled or poached.

Dinner, noon. A mutton chop without fat, broiled; well-boiled cauliflower or French beans, according to season; a thin slice of well-toasted bread; half to one wineglassful of sound sherry, diluted with twice its bulk of water.

Tea, 4 P. M. Same as breakfast.

Supper, 7 P. M. A breakfast-cupful of beef-tea (a pound to the pint); a thin slice of dry toast.

Or we can adopt a diet-table that I suggested in a lecture before the University Training-School for Nurses, which is that of the North-eastern Hospital for Children, London:

	Milk Diet.	Fish Diet.	Full Diet.
Breakfast, 7 A. M. .	Milk, $\frac{1}{2}$ pint; bread, 2 ounces, with butter.	Milk or cocoa, $\frac{1}{2}$ pint; bread, $2\frac{1}{2}$ ounces, with butter.	Milk or cocoa, $\frac{1}{2}$ pint; bread, $2\frac{1}{2}$ ounces, with butter.
Dinner, 12 M. . . .	Milk, $\frac{1}{2}$ pint; rice or other milk pudding.	Fish, boiled, $2\frac{1}{2}$ ounces; potatoes, mashed, 3 ounces; bread, 1 ounce; milk pudding.	Roast, boiled, or minced mutton, or roast or minced beef, $2\frac{1}{2}$ ounces; mashed potatoes, 4 ounces, to alternate with green vegetables; bread, 1 ounce; milk pudding.
Tea, 3.30 P. M. . . .	Milk, $\frac{1}{2}$ pint; bread, 2 ounces, with butter.	Milk, $\frac{1}{2}$ pint; bread, $2\frac{1}{2}$ ounces, with treacle or butter.	Bread, $2\frac{1}{2}$ ounces, with butter, treacle, or dripping; milk, $\frac{1}{4}$ pint.
Supper, 6 P. M. . .	Biscuit (cracker) or slice of bread and butter.	Bread, 2 ounces, with butter, or cracker.	Bread, 2 ounces, with butter, or cracker.

In the more serious forms Jacobi adheres to a very strict diet. He says: "No

raw milk, no boiled milk, no milk at all in any mixture, in bad cases." In the very worst cases total abstinence is recommended by this writer for from one to six hours; afterward the following combination is allowed: Five ounces of barley-water, one to two drachms of brandy or whiskey, the white of one egg, salt, and cane-sugar; a teaspoonful every five or fifteen minutes, according to age or case.

Jacobi in his terse way remarks: "That never are the common sense and tact of the intelligent practitioner more thoroughly taxed; no printed rule ever supplies or substitutes brains."

If the appetite be capricious, these strict dietetic rules cannot of course be adhered to; we must then endeavor to supply such a variety as will tempt the appetite and check the tissue waste. If the stools show a mass of milk curds, milk must be diluted, predigested, or altogether prohibited.

The various preparations of predigested food may now be resorted to: milk, milk-gruel, milk-punch, effervescing milk-punch, beef-tea, and oysters may all be prepared in this manner. Raw beef-juice, beef-tea, consommé, chicken, mutton, or veal broth are preparations upon which we may often place absolute dependence. Farinacea as a rule must be excluded, although we occasionally have to allow a little rice pudding, tapioca, or flour-ball by way of a variety. It is somewhat odd to note in this connection that Burnet in his valuable little book on Foods and Dietaries recommends the farinaceous substances as a suitable diet in mucous diarrhœa.

Alcohol is not by any means contraindicated, and may be administered as wine-whey or a combination of milk, egg and brandy. English writers advise well-diluted light sherry or light claret.

Among medicinal agents many and varied plans of treatment have been suggested. Recently much attention has been paid to intestinal antiseptics, but it is interesting to note that so recent and reliable a writer as Osler, in his *Practice of Medicine*, considers that "we are still without a reliable intestinal antiseptic. Neither naphthaline, salol, resorcin, salicylates, nor mercury meets the indications."

This has not been our clinical experience, nor indeed has it been that of the general practitioner.

Dujardin-Beaumetz recommends the following formula as a satisfactory intestinal antiseptic:

R̄. Salol
 Bismuthi salicylatis
 Sodii bicarbonatis āā gr. cl.

Sig. Divide in capsul. No. xxx. One capsule before breakfast and before dinner.

Droixhe considers salol as a remedy easily administered and without toxic action, and ranks it among the approved intestinal antiseptics.

Carreras suggests resorcin in the following formula:

R̄. Resorcin gr. ij-vij.
 Syr. aurantii f̄3j.
 Aq. citronellæ q. s. ad f̄3iv.—M.

Sig. Three teaspoonfuls every three hours.

The same author suggests that when the child is fed exclusively upon

milk the dejecta may be very acid; in this case some such mixture should be given as—

R_x. Bismuth. phosphat. aut subnitrat. gr. xxx.
 Sodii bicarbonat. gr. xv.
 Pepsinæ gr. viij.
 Pulv. ipecac. comp. gr. j-iv.—M.
 Divide in chart. No. iij.
 Sig. One every hour or two.

Creolin has been recommended in the following combination :

R_x. Creolin ℥viiij.
 Sacchari gr. lxxv.—M.
 Divide in chart. No. x.
 Sig. One every two or three hours.

Or,

R_x. Creolin gtt. i-ij.
 Syrupi f̄j.
 Aq. menthæ piperit. f̄ij.—M.
 Sig. Teaspoonful every two hours.

Schwinz also endorses creolin.

Naphthaline may be given to young children in doses of ten centigrammes every two hours. Pure naphthaline never causes accidents even when used in large doses. It may be given per rectum in a mucilaginous mixture which will hold it in suspension but not dissolve it. Bouchard thinks naphthol is superior in its action to naphthaline.

Constipation may exist sometimes to a stubborn degree: mild saline laxatives may be exhibited, or a simple enema may occasionally be administered, and will usually cause the expulsion of large masses of mucus.

Irrigation of the stomach is generally agreed upon by all writers to be a most efficacious method of dealing with the more chronic examples of the disease. Osler speaks of it in the warmest terms in cases of the most obstinate gastro-intestinal catarrh in children. This method must be combined with the irrigation of the large bowel. The last-quoted authority states that a pint will thoroughly irrigate the colon of a child aged six months, and a quart that of a child of two years. When the temperature is high, ice-cold water may be used for this purpose.

Booker has had a large experience in stomach-washing. His apparatus is the one proposed by Epstein. A soft Nélaton's catheter, No. 8, 9, or 10, is attached by a short glass tube to a common rubber tube two feet long, with a 2 ounce (62 grammes) glass funnel fitted into the distal end; a pitcher containing a half-gallon (2 litres) of tepid water is placed in a convenient position.

It is only within a short time that the plan of washing out the stomach, which was inaugurated by Kussmaul for diseases of that organ in adults, has been applied to children. The difficulties connected with its application are few, and the dangers, even for the youngest and weakest infants, easily avoided. Kussmaul's apparatus for irrigating the stomach consists merely of a Nélaton's catheter, a long rubber tube, and a funnel, and this simple apparatus will accomplish all that is necessary. Escherich's apparatus has greater advantages, however, and is preferred. The time required for irrigation of the stomach is

usually four or five minutes, from half a litre to a litre and a half of water being usually required before the return flow is clear. If there is gastric or intestinal catarrh, a few drops of a 6 per cent. solution of benzoate of sodium and a few drops of tincture of opium may be given hourly after each irrigation. Irrigation is contraindicated only in very feeble children and when collapse is impending. The same apparatus is also used for intestinal irrigation, excepting that a larger and stiffer catheter, with much larger lateral opening, is employed. It may be introduced, if necessary, to a distance of 27 centimetres, and the entire large intestine washed out.

Ehring's experience in this method of treatment in 850 cases has been rapid cure in 68.7 per cent. of cases, moderate success in 14.58, failure or death in 16.73. This writer further considers that the indications for this treatment exist in all cases of intestinal catarrh. Riemschneider reports the results obtained in 140 cases by this method, and is favorably impressed with the results obtained by washing out the stomach with Escherich's apparatus; he follows the irrigation of plain water by an irrigation of a 3 per cent. solution of benzoate of sodium. Of these cases a quickly favorable result was obtained in 89, a slowly favorable one in 31; in 20 the result was fatal.

Seibert in treating 1404 cases of gastro-intestinal catarrh used stomach-washing in 521 cases, and states that the results were most gratifying both in stomach- and bowel-washing.¹

Von Ziemssen recommends cutaneous electrization of the stomach with very large electrodes, for half an hour before meals. This treatment is supplemented by faradizing for a short time with the wire brush the skin of the abdomen, cheek, and back. Massage of the stomach and intestines is also of value, although of less importance than electricity.

Electrization of the intestines is accomplished with large electrodes, one occupying the entire abdominal surface, the other the entire dorsal surface; and the electricity must be of increased intensity, owing to the great size of the electrodes. The subjective results of this treatment are increased appetite and loss of abnormal abdominal sensations.

When the excretion of mucus is excessive the alkalies will assist materially in arresting its secretion: we usually select the bicarbonate of sodium; this may be combined with twenty-drop doses of tincture of myrrh, as suggested by Smith, or the powdered myrrh which Maxson speaks so highly of, given in divided doses of from 9 to 12 grains a day, either in capsules or with mucilage of acacia, glycerin, and liquorice. Podophyllin and aloes are much lauded

¹ Dr. W. Soltan Fenwick cites the dangers of washing out the stomach: 1. Convulsions and tetany. Probably because, in a case predisposed to convulsive seizures by the chronic absorption of certain morbid products from the dilated stomach, the irritation of a gastric tube may constitute an efficient exciting cause. 2. Syncope and sudden death. Any sudden alteration in the gastric pressure can, in certain cases, bring about a reflex condition of shock. 3. Perforation. The using of a gastric catheter for the purpose of investigating the chemical contents of the stomach in cases of acute gastric ulcer is a useless and mischievous procedure. 4. Hæmorrhage. Danger may arise from a too rapid evacuation of the contents of a dilated stomach. 5. Injury to the œsophagus or to the walls of the stomach. 6. Poisoning. From the use of antiseptics through the tube. Cases are cited illustrating each division. He concludes that the stomach is washed out for all sorts of symptoms, some of which are manifestly not to be benefited by this procedure. And in cases in which it fails to do good it is likely to be productive of harm in removing products of digestion whose manufacture has caused the stomach a considerable amount of labor. The indiscriminate use of this method in every case of disordered digestion will prove to be a curse rather than a benefit, and will eventually throw discredit upon the whole method of treatment.

Booker says stomach-washing is contraindicated in children affected with heart disease, serious bronchitis, or pulmonary trouble. If the tube continues to excite vomiting and strong resistance, it is doubtful if advantage follows its use.

by the English writers; our preference has been for some of the milder laxatives. We have obtained good results from the following combination:

R. Pulv. rhei 3j.
 Magnesii carb. 3iij.
 Pulv. zingiber. 3ss.
 Elixir simp. q. s. ad f3viiij.—M.
 S. Teaspoonful night and morning for child of five years.

Some cases do well upon the acids, nitric, hydrochloric, or nitro-muriatic. Strychnine, ipecacuanha, and gentian in pill is sometimes a happy combination.

Belladonna, Dover's powder, quinine, subnitrate and subcarbonate of bismuth have all been suggested. Quinine may be given in two-grain suppositories combined with a sixth of a grain of opium, as suggested by J. C. Wilson.

When the gastro-intestinal tract is in condition to receive it, iron becomes a valuable adjunct: we select either the tincture of the chloride combined with nux vomica and dilute phosphoric acid, or the dried sulphate of iron with aromatic syrup of rhubarb. Arsenic, copaiba, bromide of potassium, turpentine, cod-liver oil, oxide or nitrate of silver by mouth or by high injection into the bowel, chloride of ammonium, sulphate of zinc, bichloride of mercury, chlorate of potassium, oxide of zinc, blisters, nux vomica, ergot, are among the drugs recommended by various writers. Gold has been suggested as follows:

R. Auri 20 grammes.
 Mellis 125 grammes.—M.
 Sig. One coffeespoonful in the morning and two in the afternoon.

Antiquedad states that hydrotherapy, sulphate of quinine, chlorate of potassium, and revulsion are the means which will be found most efficient in the treatment of intestinal catarrh in children.

It is quite useless to order cod-liver oil while the alimentary canal is covered with mucus; when we have modified the mucous discharges, oil then becomes a valuable drug. These children, however, cannot assimilate large doses.

Much is to be gained by a residence in a suitable climate. We can formulate no rules, however, as to the locality to be chosen; each case is a rule unto itself. My practice has been to leave the matter of selection of a climate to a great extent to the patients themselves, with, however, a promise that the locality must be such as to permit of an almost constant out-door life, the greatest number of clear sunny days, and the least variability of thermometric range. It must also be understood that the patient will spend several years at the place of selection.

DIARRHŒAL DISEASES.

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THERE are many difficulties in the way of a satisfactory classification of the diarrhœal affections of infancy. The gravest symptoms in the most speedily fatal cases are often accompanied by the most superficial lesions; while, on the other hand, symptoms so mild that no anxiety is awakened may result from marked and extensive pathological changes. Cases which are apparently identical clinically often reveal diverse lesions. It is therefore apparent that the pathological alterations do not form a suitable basis of classification. The variations from the normal condition found after death are dependent more upon the length of the continuance of the diarrhœa than upon the primary exciting causes. The majority of cases of infantile diarrhœa which continue for four days or longer might be designated, in a classification founded upon morbid anatomy, as entero-colitis, and, moreover, the extent of the inflammatory changes is measured largely by the duration of the diarrhœa. In cases terminating fatally within four days, in previously healthy children, even the superficial epithelium may be normal, while in other of these cases there may be some desquamation of this layer. The cases which terminate fatally after from seven to ten days usually show more marked inflammatory changes. The mucous membrane is swollen, the villi are prominent and purplish, and the solitary and agminated follicles are congested and projecting. In more protracted cases the inflammatory process involves the deeper layers, and ulcerations in every degree, from the most superficial to those extending down to the muscular coat, may appear.

It would be as unscientific to attempt a classification of the diarrhœas of infancy founded upon pathological anatomy as it would be to designate acute, subacute, and chronic arsenical poisoning as desquamative, catarrhal, and ulcerative gastro-enteritis.

Having thus discarded all classifications founded upon morbid anatomy, what shall we select as a basis for the differentiation of the various forms of diarrhœa in infancy? The fundamental object in any classification must be to enable the physician to treat his patient most successfully. The giving of names to diseased conditions enables us to group, systematize, and most advantageously use the information which we may possess, or may in the future acquire, concerning the etiology, symptomatology, and treatment. Certainly in the class of diseased conditions now under consideration a classification founded upon etiological factors will be of greatest service in treatment. But the question which arises here is this: Do we at present know enough of the causes of these diseases to attempt a classification based upon etiology? In answer to this I reply that, while there is yet much to learn on this point, I propose to offer a provisional classification founded upon what I believe to be the most important factors in the causation of the diarrhœas

of infancy, because I believe that such a classification, imperfect as it must at present be, will be of greater service to the practitioner than one based upon the morbid anatomy, which, as we have seen, is determined more largely by the duration of the diarrhœa than by the nature of the exciting cause.

In attempting this classification we have the following facts to aid us: (1) Some of these diarrhœas are independent of seasonal influence. They occur as frequently in winter as in summer, while the prevalence of other forms is so plainly limited to the hot season that they are now quite universally designated as "summer diarrhœas." (2) Those which are apparently independent of seasonal influence do not differ from similar diseases in adult life, save in the greater susceptibility of the infant and in the greater delicacy of its organization, thus rendering the disease of more serious import in the child than in the adult. On the other hand, the so-called "summer diarrhœas" are so generally limited to the first two or three years of life that they may be regarded as peculiar to that age.

Improper or excessive feeding, acting upon the delicate organization of the child's digestive apparatus, may cause diarrhœa even when there are no toxigenic micro-organisms present. A small quantity of some indigestible substance in the intestines may increase the peristaltic movements and lead to frequent stools. Taube and Escherich have shown that in the young child stomachic digestion is of less importance than intestinal digestion, and that the stomach is more of a receptacle into which the milk is received for coagulation than a digestive organ; thus we have the most favorable conditions for the growth and activity of the bacteria which are introduced with the food. The same investigators find that the younger the child the less active is digestion in the stomach, and that in this organ the milk is coagulated and passed through the pylorus undigested. Hammarsten has shown that this is the case in puppies and young rabbits, and Hofmeister and Tappeiner showed that the stomach does not absorb soluble substances as rapidly as does the mucous membrane of the small intestines. Zweifel states—and in this he is supported by Hammarsten—that the proteolytic activity of the pancreatic juice is relatively well developed in the new-born. The absorption of fats is dependent upon the pancreatic juice and the bile, and the teaching of Frerichs, that the milk-sugar is absorbed from the stomach, is now known to be erroneous. This constituent of milk, as has been shown by Dastré, is digested by a ferment found in the mucus of the small intestine. These experiments convince us that the digestion of milk by the infant is nearly, if not quite, altogether accomplished in the small intestine, and explain why indigestion in the infant induces diarrhœa.

The diarrhœa which results from temporary indigestion will be described under the title of *Acute Intestinal Indigestion*.

The continued ingestion of material indigestible in character will produce inflammatory processes leading to pathological lesions, and this condition will be considered in this paper under the head of *Chronic Intestinal Indigestion*.

It must now be admitted that the so-called "summer diarrhœas" of infancy are due to the growth and multiplication of bacteria and the formation of chemical poisons by these low forms of vegetable life. Since these harmful organisms are, in the great majority of cases, taken into the body in the milk which constitutes the sole or chief food of the infant, I will describe the symptoms and lesions due to these causes under the title of *Milk Infection*, and this will be subdivided, according to the severity and duration of the symptoms, into *Acute* and *Subacute Milk Infection*.

I would prefer the term "milk-poisoning" for the last two of these forms

of diarrhoea, but, bearing in mind the fact that "milk-poisoning" has long been used to cover another affection, I have been debarred from using it. There are, moreover, certain advantages in the adoption of the words "milk infection." These bring out more prominently the part played by bacteria in the causation. I wish to positively deny that I have been led to drop the old nomenclature and adopt a new one for the sake of introducing a novelty. I believe that the advance in our knowledge of the causation of these diarrhoeas justifies the change, and that the use of the terms here suggested will, in the first place, give the physician a better idea of the cause and nature of the trouble with which he is dealing, and, secondly, it will tend to make parents more attentive to the character of the food supplied their children.

This simple classification will, as a whole, I believe, be of most service to the practitioner, and the object of this paper is to aid the physician in treatment, and not to instruct the pathologist in morbid anatomy. It must not be supposed, however, that the writer believes that this classification is perfect, or that a diarrhoea originating in one of the above-mentioned causes may not be influenced by other etiological factors. A child with a simple irritative diarrhoea is by no means immune to milk infection, and every physician knows that the intestines of an improperly-fed child furnish the best-known culture-tubes for the growth of certain harmful bacteria. For these reasons the prognosis in a case of intestinal indigestion will be influenced by the greater or less probability of there being engrafted upon this abnormal condition the more serious element of bacterial poisoning.

ACUTE INTESTINAL INDIGESTION.

Synonyms.—Simple diarrhoea; Irritative diarrhoea; Mechanical diarrhoea.

The number of cases of this disease is large, but, unfortunately, the physician is not frequently consulted concerning them until they have become chronic or until the supervention of bacterial poisoning renders the symptoms more grave and excites alarm. The idea that frequent stools are beneficial during teething has led to neglect of these cases, and has been an important factor in increasing infantile mortality. The prompt recognition and treatment of acute intestinal indigestion are most valuable prophylactic measures against the more serious intestinal disorders. Measured by the good which can be accomplished by proper treatment, this disease is second to none of the diarrhoeal affections in importance.

Etiology.—Excessive feeding is a frequent cause of intestinal indigestion. Children fed artificially are more likely to be overfed than those nursing from the breast, for two reasons: In the first place, the supply is not so easily exhausted, and, in the second place, the child obtains the food more easily; indeed, the milk is often poured into the child's stomach *ad nauseam*. To these might be added the fact that the child is often given the nursing-bottle when the busy mother would not stop to nurse it herself. Again the system needs so much water, and too many mothers and nurses seem to be wholly ignorant of the fact that a babe might relish a little water at times. The overloading of the stomach throws upon the digestive organs more work than they can do, and the undigested portions act as foreign bodies.

Improper feeding is another fertile source of mischief. This is not the place to discuss infant-feeding, and readers are referred to the special section upon that subject. It may be remarked, however, that the custom of giving the babe a taste of various things on the table is a pernicious one. The milk of the healthy mother contains all the nourishment needed by the nursing

infant, and should constitute its sole food at this period of life. But, unfortunately, the mother is not always healthy, and she may on account of sickness, excessive menstruation, or other causes be unable to supply the demand either in proper quality or quantity. In these cases the knowledge of the most intelligent physician is often found to be too limited.

The cause of the imperfect digestion may be in the child itself. It may have tuberculosis or some other wasting disease, or the digestive organs may be functionally incapacitated by some temporary ailment. The employment of predigested food may be resorted to for the time, but its continued use is not wise. The digestive organs, like all the organs of the body, are enfeebled if relieved of their physiological duties. The too rapid absorption of peptones may be harmful, and physiologically it is questionable whether proteids which have been completely converted into peptones are ever largely utilized in the body in building up tissue. It is probably fortunate that in the great majority of instances artificial digestion is incomplete and the supposed peptones are actually albumoses.

Symptoms.—Restlessness, flatulency with abdominal pain, and sometimes vomiting, are the first symptoms of this form of diarrhœa. Then frequency of stool, often accompanied by griping pain, follows. The appearance and other physical characters of the discharges vary with the severity and continuance of the attack. At first they appear quite normal, and their frequency is the only thing to attract attention. Then they become more watery, but are not mucous, as they are when the disease becomes chronic and inflammatory, nor serous, as they often are in acute milk infection. The stools are sometimes green, and this may give rise to alarm, but this color is often due to trivial causes, and too much importance has sometimes been attached to it. After a free discharge the child becomes less restless, and may fall into a quiet sleep, from which, however, it is soon aroused by abdominal pain, which continues until the bowels are again relieved. A few hours of this pain tells upon the features; the countenance becomes pale, and its continuance for a few days lessens the rotundity of the limbs and makes the muscles soft and flabby. If the intestinal irritation be severe, convulsions may occur. Elevation of temperature is seldom observed in this form of diarrhœa, or if it does appear it is evanescent. The pulse is accelerated during the paroxysms of pain, but is usually normal during the intervals. Thirst is an accompaniment, and may be great when the stools are frequent and watery.

Prognosis.—This form of diarrhœa is not in and of itself fatal. Unless the cause of the irritation be removed, inflammatory processes are induced in the intestine, and a chronic diarrhœa results, or bacterial invasion, finding favorable soil, may speedily develop an alarming condition.

Treatment.—The prompt and judicious treatment of this form of diarrhœa is in the majority of instances highly satisfactory. The administration of all food should be forbidden for a number of hours. The exact period of this prohibition may vary with the symptoms in the individual case, but, as a rule, twenty-four hours will not be too long. The child will be restless and will cry from thirst, which should be provided for by suspending bismuth subnitrate in sterilized water, from two to five grains to the drachm, and ordering that this be given in doses of a teaspoonful or more every hour when the child is awake. The undigested food remaining in the intestines should be removed, and the best agent for the accomplishment of this purpose is castor oil, a teaspoonful of which should be given to a child one year of age. Some physicians prefer rhubarb (one to two drachms of the syrup), and others recommend magnesium sulphate, but I am sure that there is nothing which is more certain

and pleasant in its action than castor oil. It may be asked whether or not the administration of the laxative is regarded as essential in every instance. I have seen many children improve rapidly without it. In these the irritating substance has been swept out of the intestines by the diarrhoeal discharges, and a small dose of opiate is all that is needed; but it is impossible to tell in a given case whether this fortunate removal has been accomplished by unaided nature or not, and the more certain method is to administer the laxative.

After the laxative has had its effect, earlier if there be great pain, an opiate in very small doses, to be repeated, if desirable, after each evacuation, is generally beneficial. The opiate may be given in the form of the tincture, the deodorized or the camphorated tincture. The custom of introducing opium into compound prescriptions ordered for children is to be condemned. It is a common practice with many physicians to write a prescription containing an opiate, bismuth subnitrate, pepsin, and chalk mixture. The pepsin is useless, because the administration of food has been prohibited, and it cannot have any digestive effect upon that which is already in the intestines. The syrup in the mixture may ferment and be harmful, and the chalk is without value, while the bismuth should be given more freely than the opiate. For these reasons the opiate should not be incorporated in a mixture, but should be prescribed by itself; and this holds good whenever opium is employed in any form of diarrhoea in infants. I have said that the dose of the opiate should be small—simply enough to allay the abnormal peristalsis of the intestines. From five to ten drops of the camphorated tincture or a half minim of either of the other tinctures will usually suffice for a dose for a child one year of age. After twelve hours of this treatment the condition of the child will usually be found to be much improved, but the diarrhoea will return as soon as the improper feeding begins. It is well to order the continuation of bismuth subnitrate at longer intervals for some days, and the physician must give his attention to the character of the food, which must now be resumed. He must endeavor to ascertain wherein the feeding was at fault, and thus avoid a repetition of the trouble. If the child is nursing and the harm has come from the giving of additional food, such addition must be forbidden. If the mother's milk is at fault, and if this cannot be improved, the selection of a good wet-nurse is the best thing that can be done. If neither of these is practicable, or if the child has been artificially fed, the selection and preparation of the best food suited to the case must be undertaken. For aid upon this point the reader is referred to the section on infant-feeding.

CHRONIC INTESTINAL INDIGESTION.

Synonyms.—Chronic diarrhoea; Chronic irritative diarrhoea; Chronic intestinal catarrh; Chronic entero-colitis.

Etiology.—Chronic intestinal indigestion, with consequent diarrhoea, is a common affection of infancy. The undigested food ferments, and the products of this fermentation, acting as irritants upon the sensitive mucous membrane, induce a catarrhal condition which is most marked in the ileum and colon, where ulceration not infrequently results. All this may occur without the aid of toxicogenic germs, and probably without the intervention of any adventitious bacteria whatever, since those normally present are capable of accomplishing these results when digestion is arrested or markedly retarded. Chronic intestinal indigestion may occur at any season of the year, but it becomes of more serious import during the hot months, when toxicogenic germs abound and the chances of their invasion are greatly increased. It is self-evident that

this affection is more common among those infants artificially fed than among those who draw their sole and sufficient nourishment from the breast of a healthy mother. It is equally plain that it is most prevalent among those suffering from debilitating and wasting disorders, either inherited or acquired, such as syphilis, tuberculosis, rickets, or chronic broncho-pneumonia; and among those who have had their vitality impaired by an acute infectious disease, such as pertussis, scarlatina, or measles. Children who suffer from neglect, insufficient clothing, and exposure to cold are also prone to this affection. Some children seem to be born with an inability on the part of the intestines to properly digest and absorb food. I have seen such a child weighing less when sixteen months of age than at the time of birth, and yet recovery resulted, and the child, now eight years of age, weighs as much and is as robust as the average. Frequent attacks of acute indigestion lead to the chronic form, though it is probably true that the majority of cases of chronic indigestion develop insidiously and without any marked preliminary acute attack.

Symptoms.—The discharges from the bowels become, as a rule, gradually more frequent, increasing from one to two, to from four to six or more per day. The child usually becomes nervous, fretful, and fails to sleep well. Flatulency is a more or less marked symptom, and when great the distention of the bowels may cause severe pain. The stools are generally quite characteristic in certain particulars. In consistency they may be semi-solid or more watery, or they may vary in this respect from time to time. The odor is quite invariably distinctly offensive. So marked and common is this that the stools are frequently designated as putrid. The presence of undigested food is indicated by the color. Lumps of coagulated casein and masses of unchanged fat may be seen. With the progress of the disease and the development of inflammatory changes, mucus appears, pus may be detected with the microscope, and, when hard lumps are present, they may be streaked with blood. The color will vary with the kind of food and the extent to which it fails to digest. Pale, putty-like stools are common, while the presence of a large amount of fat may render the excretions gray or even white. The green stools are quite common in this affection, and in some instances at least this coloration is due to the growth of chromogenic bacteria. There are likely to be periods of exacerbation, when the number of evacuations becomes much greater and their consistency thinner and more watery. At these times the pain usually becomes more severe, and fever, with vomiting and increased restlessness, makes the case more alarming. The diarrhoea, more or less marked, may continue for weeks. In rare instances the increased frequency of the discharges may be borne by the child for a long time in a surprising manner. The rotundity of the limbs is not lost, and the infant may not only hold its own, but may gain slightly in weight. Such cases, however, make the exceptions. Usually the child loses day by day. Emaciation becomes marked, the muscles of the limbs and the trunk melt away, and the head appears by contrast to be abnormally large. The gradual loss of substance and strength may end in exhaustion and death. However, this is not common, death in the majority of instances resulting not from the disease itself, but from the intercurrent of milk infection.

In cases terminating favorably recovery is usually a slow and gradual process, liable to many partial relapses. The child becomes less fretful and gives less evidence of pain. The stools decrease in number, and become more like the normal in form and color. The putrid odor is likely to be the most persistent evidence of the diseased condition.

Throughout the course of a chronic intestinal indigestion it often happens that the appetite is unimpaired. The child, while it is losing weight and after

it has been reduced to a mere shadow of its former self, may take more food than it did when well. It has often been observed that while such a child does not give any evidence of craving food, and while its restlessness is not increased by prolonging the intervals between feeding, it readily and, possibly, voraciously swallows any food offered; and it may seem that the larger the quantity of food taken, the more rapidly do the tissues melt away. Indeed, this is not altogether a merely apparent thing; it may be a reality. When the food is not digested, excessive feeding increases the irritation, deepens the inflammatory processes, multiplies the number of stools, draws upon the vital resources, and hastens the period of exhaustion.

The stomach often remains surprisingly free from involvement in this affection, and vomiting seldom occurs save during the exacerbations already referred to. The tongue is usually dry and red, though it may be covered heavily with a yellowish or brownish coat. Thrush and follicular stomatitis are not rarely seen, and the teeth may rapidly decay. These are, however, by no means constant symptoms. The vexatious cases of prolapsus ani in infants are most common among those suffering from chronic intestinal indigestion. The general vitality of the little patient is often so low that the replaced bowel is not retained, and when it becomes inflamed and swollen it may cause great pain. The skin with which the discharges come in contact may become highly inflamed, and, unless attention be given to frequent changes and the employment of protective powders, the inflammatory process may lead to ulceration.

The temperature is usually normal, though it may be elevated during the periods of exacerbation. A subnormal temperature persisting for some days is an alarming indication, and is usually followed by death. However, in cases of marked debility and exhaustion the extremities are generally cool, and need warm clothing and at times the application of artificial heat. The pulse becomes weak, and the respiration is often irregular and shallow. The ankles may become oedematous, and this condition does not necessarily imply nephritis, though structural changes in the kidney, with albuminuria, may occur.

Diagnosis.—The history of the case and careful inspection of the stools, which should always be made, will seldom leave any doubt in the mind of the intelligent physician concerning the correctness of his diagnosis. There is one point, however, which should always be considered in reaching a correct estimate of the nature and gravity of the individual case. I refer to the necessity of a careful examination of the child in every part of its anatomy. If attention is given exclusively to the bowels, important conditions may be overlooked. In some instances—and the number of these is not small—the failure of the digestive organs to perform their functions properly is due to the existence of some constitutional disease and to the effects of poisons generated in such an affection. On the other hand, the wasting which follows long-continued intestinal indigestion renders the child highly susceptible to the invasion of specific germs, and especially to those of tuberculosis. The diagnosis must therefore embrace any constitutional coexistent affection. Otherwise the physician is likely to be led astray in his prognosis and treatment.

Prognosis.—This will be influenced largely by the parentage of the child, by the cause of the indigestion, by the duration of the disease, by the season of the year, and by the presence or absence of constitutional disease. In some families the children are prone to digestive troubles; especially is this true when one or both parents are tuberculous or syphilitic. This is also likely to be the case when a child is born to very youthful parents. If the cause of the indigestion can be traced to some special error in diet, the chance of curing

the disease after the removal of the cause is, of course, greatly increased. The greater possibility of the supervention of milk infection leads to a less hopeful prognosis when this form of indigestion occurs during the hot months; and the coexistence of chronic broncho-pneumonia, tuberculosis, scorbutus, syphilis, or rachitis may render temporary improvement doubtful and permanent recovery impossible. Still another and most important subject to be considered in forming a prognosis is the sanitary surroundings of the patient and the probability of securing improvement when needed.

Treatment.—So long as the cause of the indigestion is unknown, the treatment is likely to be wrongly directed and ineffective. Some error in diet is suspected. If the child nurses, does it obtain all its food from this source? If the answer to this be in the affirmative, then the health of the mother must be investigated. Is she pregnant? is she exhausted by excessive menstruation, prolonged lactation, by care and loss of sleep, or by some constitutional disease? If any of these be demonstrated to be the real cause, the employment of a suitable wet-nurse, when such a rare and valuable aid in treatment can be found, is the best thing that can be done. However, it is far better to take the chances with artificial feeding than to trust the child to a dishonest, vicious, or diseased wet-nurse. I have known of more than one instance of the children of respectable parents contracting syphilis from such a woman. When the infant is artificially fed, it is not enough for the physician to merely inquire about the character of the food, but he must know how it is prepared and in what quantities and how frequently it is administered. The source of the food may be exceptionally good, but if it is kept in unclean vessels, in a contaminated atmosphere, or if it is administered in excessive quantities, the doctor's drugs will be of little service until the fault is discovered and removed. The physician who depends solely upon his prescriptions, and neglects the more important matter of diet, will not have reason to congratulate himself upon the success of his treatment. The child will often improve and gain in flesh when the quantity of its food is diminished. When the stools contain lumps of coagulated casein and masses of fat, or when they are acid from the fermentation of the sugar of milk, it is best to wholly discontinue the use of milk for some days and feed the child solely upon meat broths and egg-albumin. On the other hand, if the stools be alkaline and putrid, barley gruel, rice-water, and solutions of dextrin or soluble starch obtained by roasting or boiling wheat flour, may be used. As a rule, the indigestion is confined to the inability of the digestive fluids to act upon either the carbohydrates or the proteids. If the trouble lies in the former, the stools are likely to be acid and the formation of gas in the intestines marked. In such cases a diet consisting exclusively of proteids should be tried and continued, unless it should prove positively harmful, for three or four days, and if beneficial effects follow it may be longer continued. Proteid indigestion is likely to produce fetid, alkaline stools, and a diet of carbohydrates will prove beneficial. I do not claim that any absolute rules can be founded upon the above-mentioned facts, because fermentation of one of these food-constituents naturally and necessarily prevents the complete digestion of the other; but I do hold that the physician gains no information by continuing a mixed diet, and, although he may be in error in his first trial, he has made, as it were, a physiological test, and he is now better prepared to treat the case rationally. Many physicians recommend the employment of artificially digested milk, but my experience has led me to prefer the selection of an exclusive diet of either carbohydrates or proteids; and by this I do not mean the employment of halfway measures, but the exclusion of one of these food-principles should be complete. Moreover, there are, as I have stated,

grave physiological doubts about the capability of the organism to utilize peptones in the repair of wasted tissue.

The physician must never lose sight of the fact that chronic intestinal indigestion is accompanied and may be caused by lowered vitality and general loss of tone. Tonics are indicated, and the best of these is an abundant supply of pure, fresh air. Removal from the crowded city and its contaminations to the better air of the country, and especially to that of the mountains, is often of the greatest service, and should be urgently recommended to parents who are able to provide for such a change. Arsenic and *nux vomica* may be used, but they are poor substitutes for fresh air and improved sanitary surroundings. Alcohol in the form of port or sherry is often advantageous, and cod-liver oil is of service in protracted cases.

The occasional administration of laxative doses of castor oil or from two to three grains of calomel will be of service.

Opiates are to be avoided as far as possible, and are never indicated save in the painful exacerbations which may occur.

Much has been written concerning the use of intestinal antiseptics, but only a few of these are of any real value. The same is true of astringents. Bismuth subnitrate has both antiseptic and astringent properties in a mild degree, and of all such drugs it has best preserved its reputation. It should be given in large doses, fifteen grains or more, six or eight times per day, and, as in acute indigestion, it should be kept free from combination with opiates. Sodium salicylate and salol in some cases seem to be of benefit.

The lesions in the small intestines are best reached by the administration of the large doses of bismuth subnitrate, while those of the large intestine are most successfully treated by enemata. These should be employed three or four times per week. First, the bowels should be irrigated with warm water containing a little castile soap until they are completely emptied. This must be thoroughly done, and in order to secure this thoroughness the physician must either do it himself or trust it only to a trained nurse or assistant. The hips should be elevated, and a large-sized flexible catheter attached to a fountain syringe should be passed into the colon. The passage of the catheter will be facilitated by allowing the water to flow at the time. From three to four quarts of water should be used, the excess returning by the side of the tube. After the large intestines have been cleansed in this manner, one-half pint of water, containing from one to two drachms of bismuth subnitrate in suspension, should be injected and left in the bowel. Instead of the bismuth, thirty grains of tannic acid may be used. The temperature of the water, both that used in the irrigation and for the injection, should be that of the body.

The possibility of the intercurrent of serious complications should always be borne in mind and place the medical attendant on his guard. The frequency with which relapses occur necessitates continued attention to the diet, sanitary surroundings, and general health of the little patient for weeks and months after apparent recovery.

MILK INFECTION.

The diarrhoeas which prevail among infants during the summer, especially in cities and among the poorer classes, produce a fearful mortality; consequently, they have given rise to much discussion concerning their nature and causation. The theories which have been advanced to explain the origin of these diarrhoeas have included nearly everything which a lively imagination

could suggest. Learned arguments have been made to show that the most important etiological factors lie in mysterious and unknowable meteorological or telluric conditions; while, on the other hand, the keen perception of a medical genius detects "that the fatality of the disease has been appreciably increased by the introduction and universal use of the child's carriage." The limit set upon the writer of this paper by the editor will not permit indulgence in an historical sketch of these varied theories, nor will it allow of any argumentative discussion. I shall have to content myself with a bare statement of those etiological factors the existence of which has, in my opinion, been demonstrated.

These diarrhœas are due to toxicogenic (poison-producing) bacteria. There is not a specific micro-organism, as there is in tuberculosis, but any one or more of a large class of germs, the individual members of which differ from one another sufficiently morphologically to be regarded as distinct species, may be present and may produce the symptoms.

Only a very brief summary of our knowledge concerning the intestinal bacteria can be given here, while the reader is referred for more extended information to the works of Escherich, Booker, Baginsky, and Jeffries. The intestinal contents during foetal life are sterile, and remain so for a short time after birth. However, within a few hours after birth bacteria find their way into the intestines. The meconium contains quite constantly two species of bacilli and a micrococcus. One of these bacilli is a long, slender rod with a bright, glistening spore, and is known as the "head-bacillus." The other appears to be identical with *bacillus subtilis*. The micrococcus is a large circular or elliptical organism. Breslau taught that this is taken in with the air which the child swallows immediately after birth, but Escherich thinks that these bacilli found in the rectum find entrance through the anus. However, these bacteria wholly disappear with the last passage of meconium.

The normal bacterial flora of the healthy nursing child is yet more limited, so far as species are concerned, the number being two—the bacterium *lactis aerogenes* and the bacterium *coli commune*. These are known as obligatory "milk-fæces" bacteria, and are constantly present. The upper part of the duodenum is quite free from bacteria. Lower down, the small intestines contain large numbers of the bacterium *lactis aerogenes*, while in the lower part of the ileum the bacterium *coli commune* appears, and grows more abundant in the colon, throughout the whole length of which this germ is found. Other "inconstant" bacterial forms are found in the large intestines of the healthy milk-fed child. Both the bacterium *lactis aerogenes* and the *coli commune* are pathogenic to some of the lower animals when injected subcutaneously. Whether either of these ever develop pathogenic properties in diseased conditions or not is a question which has been much discussed, but which cannot be considered as positively settled at present.

The contents of the intestines in the so-called summer diarrhœas of infancy swarm with bacteria of many species, and some of these produce most powerful poisons. These bacteria multiply outside of the body, and are disseminated widely and abundantly only when the atmospheric temperature reaches 60° F. or higher. This is the reason for the restriction of these diarrhœas to the hot months of summer.

The most suitable culture-medium for the growth of these bacteria is milk, and this is the food with which they most commonly find their way into the intestines of the child. A knowledge of these facts has led to the employment of the most effective prophylactic measures for these diarrhœas. These measures may be grouped into (*a*) those which prevent the contamination of milk, and (*b*) those which destroy any germs with which the milk has already

been contaminated. Since these diarrhoeas are limited to children artificially fed in whole or in part, our prophylactic measures are devoted exclusively to cow's milk. Some years ago I formulated the following rules concerning the care necessary to prevent milk undergoing these putrefactive changes:

(a) The cows should be healthy, and the milk of any animal which seems indisposed should not be mixed with that from the healthy animals.

(b) Cows must not be fed upon swill or the refuse from breweries or glucose-factories, or upon any other fermented food.

(c) Milk cows must not be allowed to drink from stagnant pools, but must have access to fresh, pure water.

(d) The pasture must be freed from noxious weeds, and the barn and yard must be kept clean.

(e) The udders should be washed, then wiped dry, before each milking.

(f) The milk must be at once thoroughly cooled. This is best done in the summer by placing the milk-can in a tank of cold water or ice-water, the water being of the same depth as the milk in the can. It would be well if the water in the tank could be kept flowing, and this will be necessary unless ice-water is used. The tank should be thoroughly cleansed each day to prevent bad odors. The can should remain uncovered during the cooling, and the milk should be gently stirred. The temperature should be reduced to 60° F. or lower within an hour. The can should remain in the cold water until ready for delivery.

(g) Milk should be delivered during the summer in refrigerator cans or in bottles about which ice is packed during transportation.

(h) When received by the consumer it must be kept in a clean place and at a temperature some degrees below 60° F.

If all the milk used in the artificial feeding of infants could be obtained and marketed with the care demanded by the above rules, milk infection would be practically unknown and the sterilization of the infant's food would be unnecessary. However, since it is impossible for the city consumer to know that the milk, which has been transported through a long distance and has passed through the hands of several dealers, has been kept from infection, the only safe plan for him to adopt consists in the sterilization of all of that which is fed to children. There is no doubt in the mind of the writer that wholesome, uninfected milk in the raw state is a better food for the infant than cooked milk. The heat of sterilization robs the nuclein of the milk of its vital properties, as can be demonstrated by experiments. But I am equally positive that it is better to feed the city child upon sterilized milk than it is to use that which, with the prevailing ignorance and carelessness of dairymen and dealers, is likely to be infected. The risk in using unsterilized milk is too great, and the question with the parent or physician is not, Am I giving the child the best food? but, Am I giving it a poison? The choice is easily made when the matter is looked at in this light.

The toxicogenic germs grow and multiply in the milk both before and after it has been taken into the alimentary canal of the child, and elaborate chemical poisons which induce the diarrhoea and other untoward symptoms. The number of these poisons is probably as great as that of the bacteria which produce them. While they may differ in the intensity of their toxic properties, all are gastro-intestinal irritants, just as we have a number of metallic poisons which act in a similar manner. Some of these poisons have been isolated and their effects upon the lower animals have been studied. Tyrotoxicon, first found in poisonous cheese, later in ice-cream and other milk-products, has been isolated from a sample of milk a part of which had been administered to a healthy child

and had caused a severe choleric form diarrhœa. This is a most potent poison, inducing severe and continued vomiting and purging with speedy prostration, and death within a few hours if the quantity administered is sufficient. Post-mortem examination shows but little change. The mucous membrane of the small intestine is bleached and softened, and possibly deprived here and there of its superficial epithelium. These are the symptoms and the post-mortem appearances in the choleric form diarrhœa of infants.

In 1890 proteid poisons were isolated by the writer from cultures of three of the toxicogenic germs found by Booker in the intestines of infants suffering from milk infection. These proteids are highly poisonous, and when injected under the skin of kittens or puppies they cause vomiting and purging, and, when employed in sufficient quantity, collapse and death. Post-mortem examination shows the small intestine pale throughout and constricted in places. The heart has been invariably, so far, found in diastole and filled with blood.

A small amount of the proteid from bacillus *x*, dissolved in water, was injected under the skin on the back of a kitten. Within one half hour the animal began to vomit and purge, and death resulted within eighteen hours. The small intestines were pale, contracted in places, and contained a frothy mucus. The stomach was distended with gas, and contained mucus stained yellow with bile. The liver was normal, the spleen and kidneys were congested, and the heart was distended.

Another kitten was treated with the proteid from bacillus *a*, dissolved in water. The vomited and fecal matters in this case were green. The animal died after fifteen hours, and presented appearances practically identical with those mentioned above.

A third kitten was treated with some of the proteid from bacillus *A*, suspended in water, and presented substantially the same symptoms and post-mortem appearances.

Concerning the amount of one of these proteids necessary to produce a fatal result in the animals experimented upon the following tests were made: Fifteen milligrammes of the dry proteid from bacillus *a* was injected under the skin of the back of a guinea-pig: This caused death within twelve hours. Of two kittens treated with fifteen milligrammes of the *a* proteid, one died after forty-eight hours, and the other recovered after two days of vomiting and purging. Two puppies of about five pounds weight had each forty milligrammes, and after serious illness of two days speedily recovered. During these two days of vomiting and purging these dogs were constantly shivering as with cold, but the rectal temperature stood at from 102.5° to 103.5° F.

Baginsky and Stadthagen have isolated from cultures of the "white liquefying germ" obtained by the former from diarrhœal stools a poisonous proteid which produces in mice, after about five hours, slight dyspnœa. The coat becomes rough, the animal sits with drooping head, and when forced to move does so sluggishly, but without any evidence of paralysis. The marked apathy increases, and death results after two or three days. Section shows an infiltration about the place of injection, congestion of the spleen, liver, and peritoneum. The intestine is hyperæmic throughout its entire length, and its upper portion contains a reddish-brown fluid. The same bacterium produces a poisonous base.

With our present knowledge of infected milk and the chemical poisons which may be generated therein the causation of the summer diarrhœas in infancy has been divested of the mystery which formerly obscured our views. Uninfected milk improperly administered may, as we have seen, cause intestinal

indigestion, and thus prepare the way for milk infection; but it can never directly induce the severer forms of diarrhoea which make infantile mortality so alarmingly great. The relation between these forms of diarrhoea may be likened to that between catching cold and infection with tuberculosis. The popular idea is that tuberculosis originates in frequent colds, but the physician knows that this is not true, and that the only causal relation between the two is that which grows out of the lowered vitality, lessened resistance, and greater susceptibility. If parents were willing to pay for wholesome, uninfected milk half the fancy price which they readily give for some prepared baby food, their children would be better nourished and disease among them would be less frequent.

ACUTE MILK INFECTION.

Synonyms.—Cholera infantum; Choleriform diarrhoea.

Etiology.—Fortunately, this form of milk infection is not so common as those of a milder type. It practically never occurs among children fed exclusively from the breast. The exceptions to this, if there be such, must arise from the introduction of powerful toxicogenic germs into the alimentary canal in some unusual manner. There are recorded cases in which, after a night of debauch, the milk of a wet-nurse has proved intensely poisonous to the child. It may possibly happen that an infant creeping about a filthy apartment, and investigating every object upon which it can lay its hands, by the sense of taste or by sucking its dirty fingers, may thus infect itself. It may also happen that a like misfortune may result from bacteria taken from the exterior of the breast of a filthy mother. However, as stated above, these are unusual methods of infection, and the rule holds good that choleriform diarrhoea is limited to the artificially fed.

The diligent researches of able bacteriologists—among whom Booker and Jeffries in this country and Escherich and Baginsky in Germany deserve mention—have failed to discover a specific micro-organism in cholera infantum. Booker found bacteria belonging to the proteus group most frequent in these cases.

As has been stated, the writer found tyrotoxin in one sample of milk, the administration of which to a healthy child was followed within two hours by the development of a most violent form of this kind of poisoning. This demonstrates that the poison may exist preformed in the milk at the time of its administration. Holt has observed that cholera infantum “is most frequently engrafted upon a mild dyspeptic diarrhoea.” This is undoubtedly often the case, but it so happens that in the writer’s experience the violent symptoms have suddenly appeared in previously healthy children, and Christopher makes a similar observation. It certainly is an error to say that acute milk infection begins as a mild diarrhoea. The former may supervene on the latter, but one is no part of the other.

Choleriform diarrhoea never occurs save in the hot months of summer, at a time when poison-producing germs are most abundantly distributed. The cause is invariably in the food, and the poisons which induce the symptoms are not known to originate in any other food than milk or some milk preparation. I saw one case in a child fed upon condensed milk, and the mother noticed when she opened the can that the ends were distended by accumulated gases, and the first feeding from this can was followed by severe vomiting and purging. Bacteria were abundant in the contents of the can. Another case resulted from the first feeding from a can of a baby-food preparation. Every case of this affection is one of poisoning from the elaboration of chemical products by the growth of

bacteria in milk. There may be enough of the poison in the food at the time of its administration to develop the symptoms as quickly as they would result from the giving of a poisonous dose of arsenic, or the greater part of the toxic substance may be generated by the growth of the bacteria in the alimentary canal.

Symptoms.—No one can see a little patient suffering from acute milk infection without being deeply impressed with the similarity of the symptoms with those induced by some powerful gastro-intestinal irritant. The child, which may have been perfectly well or suffering from some mild form of diarrhoea, suddenly begins to vomit and purge. These symptoms may continue almost incessantly until death results within a few hours. The color leaves the face, and a deathly pallor spreads over the countenance. The eyes sink into their sockets, while anxiety and alarm make themselves visible in every feature. Any food-contents of the stomach are soon removed by the vomiting, but this distressing symptom continues, and mucus colored with bile is thrown off. The frequency of the vomiting is increased by the administration of food or drink. The stools at first contain formed faecal matter and undigested food; then they become more watery and copious, and at last they are composed almost solely of blood-serum. At first they are yellow or green, but as they become more abundant they lose all color. The odor is peculiar and musty. Thirty or more stools may be passed in the severer cases within twenty-four hours. So long as the stools contain undigested food they may be acid, but the serous passages are alkaline. The flesh rapidly disappears, and there is no other disease, with the exception of Asiatic cholera, in which the wasting proceeds more speedily and exhaustion results more quickly. The skin is usually cool and clammy, but the rectal temperature is elevated, usually from 102° to 104° F., and in the severer cases it may read as high as 107° or 108° before death. The pulse is weak, thready, and rapid. The respirations are shallow, irregular, and hurried. At first the child cries, then only moans, and later falls into a comatose condition, but there may be great restlessness, wild delirium, and convulsions. Thirst is usually great, and everything offered is swallowed and almost immediately vomited. The abdomen is not distended, but is usually retracted. Sometimes the vomiting and purging suddenly cease, and the parents are rejoiced at this apparently favorable turn. However, it may be but the precursor of death. The physician is not cheered by the cessation of these symptoms if the child remains in a stupor, for this is most likely to deepen into coma.

In rare instances the child quickly passes into an algid state in which the temperature is subnormal. This indicates that the amount of the poison absorbed is large and the chances of recovery are small. In these cases the child lies in a stupor, with the eyelids half open and the eyes apparently covered with a film. The angles of the mouth are retracted and the lips open. The fontanelle is depressed, the pulse weak, and the respiration irregular. The urine is scanty and there may be complete suppression.

In other cases the symptoms are not so grave as those indicated above. The stools are not so frequent and copious, and the vomiting not so incessant. The little patient may brighten up at intervals, and sufficient of the poison may be removed by the vomiting and purging to give great relief and lead to speedy recovery.

Cases of acute milk infection terminate either in death or in marked improvement within forty-eight or at the most seventy-two hours. The improvement may be rapid and complete, or it may reach a certain point and there remain comparatively stationary.

Diagnosis.—There is only one disease which presents symptoms with which those resulting from acute milk infection can be confounded. This is Asiatic cholera, and at times of the prevalence of this foreign scourge a differential diagnosis between the two cannot be made without the aid of a bacteriological study of the stools. At all other times the suddenness of the onset, the incessant vomiting, the frequent and copious watery stools, and the speedy prostration are so striking and characteristic that there can be no hesitancy in making a diagnosis. It is true that some writers have tried to confound acute milk infection and sunstroke. The points of similarity are the suddenness of the prostration and the high temperature, but in the former of these there is a difference. The prostration of sunstroke is like a lightning flash, while in milk infection it develops only after a few hours. In thermic fever there may be one or two copious discharges from the bowels, but frequent purging does not occur and the stools are never serous. The attempt to make acute milk infection identical with thermic fever arose from our former ignorance of the existence of the powerful poisons which may be elaborated in milk, and the idea does not now find any support.

Prognosis.—It is quite necessary that the physician appreciate the gravity of these cases of acute milk infection. The usual termination is in death. The physician who speaks too hopefully in the first hours of the attack is likely to find himself disappointed in a very short time. The more persistent the vomiting and purging, and the more marked the nervous symptoms, the less are the chances of recovery. If the stools become less frequent and less watery, and if at the same time the pulse grows less frequent and stronger and the nervous symptoms improve, hope may be indulged in, but in the most favorable cases there is always the possibility of a relapse into the subacute form, and so long as this continues danger is imminent. Unfortunately, the name “cholera infantum” has been made to cover all the diarrhœas prevailing during hot months, and the physician must not be led astray by the reported success of various methods of treatment.

Treatment.—These are cases of acute poisoning, and prompt, energetic treatment is demanded as truly as if the child had swallowed a toxic dose of arsenic or antimony. It is certainly true that the physician who hesitates or temporizes loses his patient.

The first thing to be done is to positively forbid the further administration of the poison. *Not a drop of milk should be given.* This is a *sine quâ non* in the treatment. This prohibition of milk must be absolute. Sterilized milk is not to be thought of, and even the breast of the mother or wet-nurse must be denied. Prepared baby foods should be thrown out of the window. The most dangerous foe with whom the doctor has to contend in the treatment is the grandmother or other good-hearted old lady, who knows just what will agree with the baby, and who persists in giving it food as soon as the doctor turns his back. The most valuable ally that he can have is a trained, conscientious nurse who will carry out directions to the letter.

The second thing to do is to remove so far as is possible the poison already in the alimentary canal. Take a lesson from nature. The vomiting and purging are attempts to eliminate the harmful substance, but, like many other attempts on the part of nature, they are ineffectual and exhausting. Wash out the stomach and intestines on the first appearance of the symptoms. Do not postpone these measures in the hope that resort to them may not be necessary. What would be thought of the physician who when called to see a person who had swallowed a drachm of white arsenic should say, “Well, the symptoms are not at present alarming; I will call around after a few hours, and if it be

necessary I will then wash out the stomach"? Acute milk infection is poisoning with a substance more powerful and deadly than white arsenic. The washing of the stomach and intestine will not exhaust the little patient half so much as the continued vomiting and purging, and the artificial measures are much more effective. The bowels should be thoroughly irrigated with warm water and castile soap, not less than a gallon of the water being used. After the large intestine has been cleansed in this manner, an injection of cool water, containing fifteen to thirty grains of tannic acid to the pint, should immediately follow. Some of the poisons formed are, as we have seen, proteids which are precipitated by tannic acid, but until the great mass of proteid in the large intestine has been removed no good can be expected from this agent. The object of the tannic-acid irrigation is to render inert any soluble poisonous proteids which may remain in the intestines after the first washing.

The stomach should be washed with warm water containing a teaspoonful of common salt to the pint. After this organ has been thoroughly cleansed, from three to five grains of calomel should be administered.

These irrigations should be repeated as soon as the vomiting or purging returns. These may appear to be heroic measures, but the strength of the patient is conserved thereby to the extent to which the vomiting and purging are allayed.

The calomel is given for its antifermentative action and in order to reach the small intestines, which are inaccessible by the processes of irrigation.

After the vomiting has been allayed by irrigation, stimulants may be given by the mouth. I prefer whiskey to all other alcoholic stimulants. Brandy, if pure, would be equally good, possibly better, but unadulterated brandy is a rare article in this country, while good whiskey is easily obtainable. The stimulant is best given in ice-cold water (the water should be boiled, and then ice-packed about the container; the ice should not be put in the water) containing 0.1 per cent. of hydrochloric acid. This dilute acid may be used at any time to allay thirst.

I agree with Holt that the hypodermatic use of very small doses of morphine and atropine (one-hundredth of a grain of the former and one eight-hundredth of the latter) may be of benefit as a heart stimulant, but the dose must not be repeated too frequently. I have feared digitaline too much to try it in these cases, nor have I employed sparteine.

When the temperature is above 103° , an ice-cap on the head is desirable, and in some instances it seems to favorably affect the vomiting. When the temperature goes up to 104° or higher, some more efficient means of reducing it should be resorted to. The use of the coal-tar derivatives for this purpose is not to be considered, and the same may be said of all drugs. Frequent sponging and friction with cloths wet with cold water may be sufficient. The friction is important on account of the coldness of the surface. When the temperature is more alarming, the child should be placed in warm water, and the temperature of this gradually lowered by the addition of ice to 85° , the child being rubbed all the while it is in the bath. It should not be kept in the bath more than ten minutes after the temperature has been lowered to the above-mentioned point. Bathing the extremities in hot mustard-water and the use of friction are beneficial in the state of collapse.

With the exception of the above-mentioned stimulants the child should have no food for twenty-four hours or even longer. Then warm meat broths, given a teaspoonful at a time, and to be discontinued if they provoke vomiting, are most likely to be borne. The absolute prohibition of milk should hold good for several days.

There is scarcely a drug which has been shown to have, or supposed to have, germicidal properties that has not been used in this disease. Among others, mercuric chloride, carbolic acid, creasote, salicylate of sodium, benzoate of sodium, salol, naphthalin, and resorcin may be mentioned. These and others may be given by the mouth and by the rectum. Much harm and no good can be obtained from them. To attempt to disinfect the alimentary canal by means of these agents is a waste of time and energy which might be given to the more rational treatment outlined above.

The diapers from children suffering from milk infection should always be disinfected, and, what is of more importance, the nurse's hands should be disinfected after she has removed the diaper.

SUBACUTE MILK INFECTION.

Synonyms.—Summer diarrhoea; Gastro-intestinal catarrh; Infectious diarrhoea; Entero-colitis.

Etiology.—This is the disease which carries off so many thousands of children in the large cities every summer. It prevails only during the hot months, when the atmospheric temperature stands above 60° F. for several consecutive days. It is due to the action of poisons generated by the growth and multiplication of bacteria. These germs are certainly more widely distributed than those which induce the symptoms described under Acute Milk Infection, but the chemical poisons produced by the former are less powerfully toxic than those of the latter. However, the milder poisons induce the greater number of deaths, on account of the greater number of individuals invaded by the germs which produce them. There are also greater variations in the symptoms of subacute cases. When the chemical poisons have been studied more thoroughly, these variations will doubtless be better understood and a more exact classification of them can be made.

Symptoms.—In the milder forms the symptoms gradually develop. The movements of the bowels increase in frequency and become more watery. They consist largely of undigested food, and contain lumps of coagulated casein and masses of fat. The color may be brown, yellow, or green, and the odor, though it may be disagreeable, has not the peculiar putrid property characteristic of chronic intestinal indigestion. J. Lewis Smith has made a microscopical study of the fæces, and has the following to say concerning them: "In addition to undigested casein, I have found epithelial cells, single or in clusters (sometimes regularly arranged as if detached in mass from the villi), fibres of meat, crystalline formations, mucus, and occasionally blood. In one instance I observed an appearance resembling three or four crypts of Lieberkühn united, probably thrown off by ulceration. If the stools are green, colored masses of various sizes, but mostly small, are also seen under the microscope."

The continuance of the intestinal fermentation sets up inflammatory processes, and the stools then contain mucus. This condition may go on for weeks, and the anatomical changes in the intestines become gradually more serious. Ulcerations may occur, especially in the ileum and colon. The general nutrition of the child becomes impaired, the appetite is not good, the tongue is covered with a white or grayish coat, and there is a gradual loss of flesh. The temperature is the best indication of the rapidity with which inflammatory changes are occurring in the intestines. There is always fever, at least during some portion of the twenty-four hours, but in the milder cases it may be so slight that it is likely to escape detection. These cases, in the earlier stages

and before marked inflammatory changes have occurred in the intestines, are often readily amenable to treatment, especially to proper change in food, and marked improvement may be produced in a short time. Other cases are more obstinate and drag on for weeks, and are likely to terminate fatally from some exacerbation, from exhaustion, or from some intercurrent disease. Children who have suffered from this slow poisoning during the summer are likely to fall victims to pneumonia the succeeding winter.

In these protracted cases there is usually more or less vomiting. This may be an early symptom, in which case it is due to fermentation in the stomach; or it may appear later when stomachic digestion is impaired by the general failure in nutrition. The vomiting is not so incessant as it is in acute milk infection.

During the progress of the protracted cases there are likely to be many exacerbations, or acute infection may result from the introduction of more virulent toxicogenic germs.

In other instances the development of these symptoms is more abrupt. The child becomes restless, and cries with pain due to distention of the intestines with gas, and there may be convulsions. Vomiting occurs early, and the temperature may rise to 103° . The diarrhoea begins, and the expulsion of the stools is accompanied by large quantities of gas. This gives relief from the pain, the nervous symptoms disappear, and the child falls asleep, from which it is soon awakened by new accumulations of gas. In these cases unaided nature is frequently successful in removing the offending contents of the intestines, and unless the administration of infected food is continued a speedy return to health may follow. Under other conditions the severe initial symptoms abate, but putrefactive processes continue in the intestines for an indefinite period of time.

Whether the symptoms come on gradually or begin more abruptly, the continuance of bacterial fermentation in the intestines leads to the development of those anatomical changes which constitute what is generally designated as enterocolitis. That the fermented intestinal contents are irritant in their action is shown by the erythema which appears on the buttocks and thighs when frequently soiled by the discharges, and which may develop into superficial ulceration of the skin. It is generally believed that the structural changes in the intestines are due to the direct action of the bacteria on the intestines, but these alterations are more probably due to the irritating action of the chemical products of the germs. The upper parts of the small intestines, the duodenum and the jejunum, are generally free from inflammatory changes, which are marked in the lower part of the ileum. This is easily explained by the fact that the contents of the small intestines accumulate here before passing through the ileo-cæcal valve. If the destructive processes in the intestinal walls were due to the direct action of the bacteria burrowing into the tissue, the explanation of the location of the catarrhal inflammation and the ulceration in the lower ileum would not be easy. Inflammatory changes in the colon are invariably present in protracted cases, and they are generally more marked than those of the small intestines, due to the fact that the intestinal contents become more irritating the longer they are subjected to the fermentative action of the bacteria. While the anatomical changes are frequently found along the entire course of the colon from the ileo-cæcal valve to the sigmoid flexure, they are most marked just above the last-mentioned point. This is again explained by the delay which occurs here in the passage of the irritating substance. The rectum is usually free from inflammatory lesions, or shows only those of the most superficial character.

The extent to which these anatomical lesions are developed depends upon the character and quantity of the irritating substances formed, but most of all upon the duration of the diarrhœa. A milder irritant acting through a longer time may cause deeper and more dangerous tissue-changes than a more powerful agent acting for a shorter time. The character and extent of these lesions may be to some extent judged by the contents of the stools. There may be much fluid mucus in the passages, and in such cases it is customary to say that the child is suffering from "catarrhal diarrhœa," or there may be lumps or clots of mucus stained with blood, and this is designated as "dysenteric diarrhœa." The presence of shreds of mucous membrane has led to the use of the term "croupous diarrhœa," and the detection of considerable pus is deemed sufficient to pronounce the case one of "follicular ulceration." However, as all of these changes may result from one and the same poison in different degrees of concentration or acting through varying periods of time, a classification based on the anatomical lesions is wholly irrational. It must not be concluded from this repudiation of an anatomical basis of classification that the physician should pay no attention to the stools. Careful inspection should be made frequently, and the statements of attendants should not be relied upon to the extent of failing to give this matter personal attention. Because one knows that his patient is poisoned with arsenic, this is no reason why he should shut his eyes to the amount and extent of gastro-intestinal irritation caused by the poison, or even to the condition of the circulation, respiration, and nervous functions. Learn all you can about your patient, and you will often find yourself even then knowing too little to effect a cure.

Complications.—Erythema of the buttocks and thighs from the irritation of the discharges is frequent, and, as has been stated, superficial ulceration may be developed and may form a very distressing complication. Thorough cleansing, the use of a mild soap, and subsequent dusting with starch or other protective powder should be advised.

Boils over the head and face often appear, and the destruction of tissue may be so deep that permanent scars are formed.

In strumous children the lymphatic glands in the inguinal region, more rarely those about the throat, may enlarge and possibly suppurate. I once saw a case in which the suppuration from the glands of the neck was so profuse that it endangered life. The urine, which was normal before the glands began to swell, contained a considerable quantity of blood, and the hæmaturia continued for more than a week. The glands were freely opened and antiseptically treated, and the child ultimately recovered completely.

In the great majority of these cases the stomach remains surprisingly free from any lesion, and this is true even when there has been frequent vomiting. In a small number some hyperæmia of the mucous membrane of this organ is found after death, and in rare instances minute ulcers have been observed.

Stomatitis is frequently a complication, and aphthous ulceration an occasional one.

Hypostatic congestion of the lungs is frequent, and a subacute bronchopneumonia is a common complication of this form of diarrhœa. It is most marked in the posterior and dependent portions of the lungs, and it often constitutes the immediate cause of death. The condition of the patient in protracted cases renders it specially susceptible to specific micro-organisms, and tuberculosis is sometimes developed.

Holt thinks that the frequency of nephritis as a complication has been over-estimated since the writings of Kjellberg called attention to it, and J. Lewis Smith doubts the correctness of generally attributing the vomiting to uræmic

poisoning. My own observation and belief support the views of these American authorities.

Diagnosis.—Subacute milk infection is distinguished from the acute form by the milder character of the former. The vomiting and purging are less violent, the temperature does not rise so high, the prostration is not so great, and the large serous stools, so characteristic of the acute form, are wanting. From chronic intestinal indigestion there may be great difficulty in making a differential diagnosis. The season of the year, the character of the food, and the hygienic surroundings must be taken into consideration. The temperature is also another valuable indication, as an elevation is exceptional in indigestion except during periods of exacerbation. From intussusception, subacute milk infection is to be distinguished by the suddenness and violence of the attack, the tenesmus and pain, the absence of fever, and the stercoraceous vomiting which characterize the former.

Prognosis.—As in the case of chronic intestinal indigestion, the prognosis will be influenced by the parentage of the child, by its sanitary surroundings, and by the period of time through which the poisoning has continued, and consequently by the extent and character of the anatomical lesions. Cases developing at the beginning of a hot summer, especially when the parents are not able to transfer the child from the crowded and possibly filthy quarter of a city to a salubrious country place, are less likely to recover than those occurring among the same classes late in the fall. The probability of relapses, when the surroundings remain unfavorable, should always be borne in mind.

Treatment.—Preventive treatment intelligently carried out would save thousands of lives annually in our large cities. The best of all these measures is that the mother should nurse the child, and the mother who allows anything short of absolute inability to prevent her doing so places the life of her child in jeopardy. Daily bathing should be practised; and again I must call attention to the desirability of having nurses disinfect their hands after they have changed the diapers of the infant. This should be done whether the child is sick or well. Reports showing that all the children in a hospital fed by a certain nurse have simultaneously developed a diarrhoea, while those fed with the same food by other nurses have remained well, are given by some writers in order to prove the contagious character of the disease. It is more than likely that these cases were due to direct infection of the food from the hands of the nurse or from the use of unclean receptacles. Soiled diapers, even those from healthy infants, should not be allowed to dry in the air which children breathe. When the mother cannot nurse her infant, the fresh, uncooked, uninfected milk of a healthy cow is the best substitute. When this cannot be obtained with any certainty, sterilized milk is the next best food from a prophylactic standpoint. Fresh air, and plenty of exercise in it, are essential to the proper growth of the child.

When we come to the curative treatment the question of feeding is one of the most perplexing with which the physician has to deal, and the writer rejoices that for the details on this point he can refer the reader to the high authority who deals with the subject of Infant-feeding in this volume. However, it is not fair to shirk all responsibility in this matter, and a brief statement of the dietetic treatment will be given.

We will assume that the child has been artificially fed in whole or in part. All milk food should be prohibited for from two to four days, possibly longer. Escherich has shown that the bacterial flora of the infant's intestines changes radically and speedily when milk is excluded from the diet. In fact, this is one of the most potent agents at our command for destroying toxicogenic germs

in the intestines. Their best culture-medium is milk, and in this they will thrive and multiply most abundantly. Exclude milk from the food, and these bacteria give place to others which, if toxicogenic at all, are less powerfully so. The proteids of the milk may be replaced by animal broths and solutions of egg-albumin, which should always be freshly prepared. The meat extracts of trade are worse than worthless in these cases. Their nutritive value is practically zero. They contain extractives which may be used as stimulants, but these are not specially indicated in the cases now under discussion. The carbohydrates are best supplied in the form of soluble starch and dextrin, obtained by boiling rice or arrow-root or by baking these or other foods rich in starches. A return to a milk diet should be made cautiously: sterilized milk should be employed, and at first in very small quantities, the greater part of the food still consisting of the articles mentioned above.

Shall the medicinal treatment be begun by the administration of a laxative? The answer to this depends upon the period in the development of the disease when the physician first sees the patient. In dispensary work, and often in private practice, the physician does not see these cases until the diarrhoea has persisted for days, possibly for weeks, and after the little one has been dosed with domestic remedies, which are practically unlimited in number and variety. If the child is seen early, give from one to two teaspoonfuls of castor oil, followed by one or two drops of the tincture of opium. If, on the other hand, the child is already exhausted from the continuance of the diarrhoea, begin at once the administration of stimulants, whiskey or brandy, and give opium in small doses, which may be repeated sufficiently to allay any pain and lessen the peristaltic action of the intestines, but never sufficiently to induce constipation. Irrigation of the intestines, as before described, should be resorted to in all cases. After the large intestine has been cleansed by irrigation, from two to three drachms of bismuth subnitrate should be suspended in from six to eight ounces of water and retained as long as possible. The irrigation of the intestines, with the subsequent injection, may be practised from two to four times per week so long as the stools remain abnormal. Tannic acid, ten to fifteen grains to the ounce of water, may be used instead of the bismuth. Irrigation of the stomach is seldom indicated—never unless the vomiting be a marked symptom. Bismuth subnitrate suspended in water or in some mucilaginous drink should be given by the mouth in quantities of one or two drachms per day.

Antiseptics are practically without value, and, as unnecessary dosing is certainly to be avoided, medication should be without them. The astringents, both vegetable and mineral, such as catechu, coto-bark, silver nitrate, and lead acetate, which are so frequently found in diarrhoea mixtures, are not only valueless when given by the mouth, but they are likely to interfere with the digestive action of the stomach, which, as we have seen, usually escapes involvement in the diseased process, and consequently they are harmful.

In protracted cases general tonic treatment is often of great value. Dilute nitro-hydrochloric acid, three or four drops in as many ounces of water, is one of the best in the list of tonics. Fowler's solution, two or three drops three times per day, may be of service, and the tincture of nux vomica has been much praised. Iron and cod-liver oil are most appropriate after the digestive disturbances have disappeared.

In the more acute forms, where tenesmus is marked, relief may be obtained by the use of suppositories containing one-fourth of a grain of cocaine. Hot applications over the abdomen may also be of value.

I must again emphasize the need of attention to the local sanitary con-

ditions in all cases of milk infection. These are of more importance than the climatic influences; and, moreover, the former can be improved, while the latter can be bettered only by a change in residence. Unhygienic surroundings tell most unfavorably upon the young child, whose organism requires time in order to adapt itself to its environment.

DYSENTERY.¹

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DYSENTERY is an inflammation of the mucous membrane of the large intestine. In retaining the term "dysentery" in the nomenclature of diseases of the intestines it is not because it is recognized as a distinct form of disease, but because it is a convenient term to express the most prominent symptoms resulting from the lesions in the colon and rectum.

There are three varieties—the catarrhal, the diphtheritic, and the amœbic.

I. CATARRHAL DYSENTERY.

This affection may be acute or chronic, sporadic, endemic, or epidemic.

Etiology.—Catarrhal dysentery may occur at any age from birth to puberty, but it is most frequent between the first and tenth year as an independent affection. Sex exerts no influence, as it occurs as frequently in boys as in girls; and the same statement is true of race; so if it happen to occur more generally among any particular nationality in a community, it must be attributed to other than racial influence. It occurs under all social conditions from the highest to the lowest, and, while it is more prevalent among the pauper and laboring classes, its severity is not tempered by high social standing. It is more frequent in the city than in the country, but occurs in the latter with as great severity as in the former. Neglect, poverty, ill-ventilated and uncleanly apartments, and insufficient and foul clothing act as predisposing causes by depressing the general resisting powers of the child. Hence it is met with more frequently among the inhabitants of the tenement-houses than among those in sanitary dwellings. The liability to dysentery is increased by such vices of constitution as tuberculosis, congenital syphilis, rickets, and athrepsia, which enfeeble the general health.

As dysentery frequently occurs during the period of the eruption of the deciduous teeth, there is a popular belief that it is the direct result of dentition. After careful observation and study of the relation of dentition to diseases of the alimentary tract in 288 infants, the author feels free to assert that neither the evolution nor eruption of the teeth was found to be an etiological factor in any of them. Similar investigations may convince the skeptical that improper alimentation, and not "teething," is the most potent etiological factor in the disorders of the alimentary tract of infants. So in order to establish a direct relation between dentition and dysentery every other etiological factor must be excluded.

The most frequent as well as the most powerful causative factor is improper

¹ At the meeting of the American Pediatric Society, held at West Point in May 1893, it was agreed to drop the term "Dysentery" from the nomenclature of diseases and substitute for it "Ileo-Colitis."

feeding. The food may be faulty in quality or quantity, or in both. Pure food may act deleteriously if given in too large quantities; and the correct amount of impure food will certainly prove injurious. It occurs most often in the artificially fed, although the nursling is not exempt. The former is not only subjected to the perils of impure or ill-prepared cow's milk, but also to the dangers of the indiscriminate use of indigestible "table-food." We frequently see a baby lying in its crib or carriage with a half-filled bottle of decomposed cow's milk, or, still worse, a concoction of some patent "infant food," lying beside him, to the foul fly-infected tip of which he applies his lips for comfort day and night. Changing the drinking-water, whether it be impregnated with harmful germs or not, may irritate the intestinal canal. The author has in his possession records of at least fifty children who undoubtedly contracted dysentery by drinking the water from an impure city well. In this instance the disease prevailed very generally among the children in an area of several blocks, but those who did not use that pump-water were almost entirely exempt. Seeds, uncooked vegetables, unripe or decayed fruit, toys, coins, and many other indigestible substances may induce dysentery by injuring the intestines in their passage through them. Weaning has been noted by some as an etiological factor, but it must be remembered that coincident with it is the introduction of artificial food—a recognized factor. Sudden changes of temperature, particularly sudden and rapid falls, or exposure to draughts of air, may chill the body and cause dysentery. It is now very generally accepted that bacteria play an important part in the production of this disease, but as yet experimentation has failed to detect a specific germ for the catarrhal form. Finally, the anatomical lesions of catarrhal dysentery vary so much that we are forced to the inevitable conclusion that no single etiological factor will cause them.

Morbid Anatomy.—The lesions of catarrhal dysentery are usually confined to the lower part of the colon and rectum, but in some cases may extend along the upper part of the colon, and even into the ileum. They are characterized by more or less intense hyperæmia of the mucous membrane, either general or confined to circumscribed areas, and there may be slight punctiform hæmorrhages into the mucosa or submucosa. The congested mucous membrane varies in color from bright-red to dark-purple, and is never uniform; it is usually covered with thick, tenacious mucus. The large intestine is usually empty, while the small is distended with gas and contains a thin greenish fluid. The mucous membrane is commonly swollen and grayish in color. The solitary lymph-follicles along the colon are swollen, sometimes to the size of a small bean, and surrounded by an area of hyperæmia. Between these inflamed areas the mucous membrane is normal in appearance. Ulceration may take place. The ulcers at first are round and superficial, but soon enlarge, two or more coalescing and forming ulcers from one-half to one inch in diameter, often exposing the muscular coat of the intestine. Their edges are everted and flattened, and they assume an irregular, serpentine, or rodent shape. Ulcerations in different stages of development may often be found in the same individual. Patches resembling pseudo-membrane may also be found. Cicatrization begins upon the floor of the ulcer, its edges being drawn toward the base. Perforation and peritonitis, which are seldom seen in children, may result from the ulcerative process extending through the intestinal coats. The liver, which is usually congested, may be the seat of multiple abscesses. The mesenteric glands are enlarged and softened and dark blue in color.

Bouchut found thrombi in the sinuses of the dura mater in 35 of the 38 children who had died of "dysenteric convulsions," and in the other 3, en-

cephalitis. Busey verified by his cases the results obtained by Bouchut. Cerebral anæmia, which is the commonly accepted cause of convulsions or death, may be found alone or coexisting with thrombosis of the sinuses of the dura mater. Busey has also observed, in a few fatal cases in very young children, œdema of the lower extremities and discoloration of the skin of the feet and legs, which he attributes to the formation of thrombi in the pelvic veins, causing venous stasis and serous transudation into the subcutaneous tissues.

The following reports of necropsies illustrate some of the principal macroscopic lesions of dysentery:

Child, aged fourteen months, great emaciation, muscles flabby, and rigor mortis deficient. *Lungs*.—Hypostatic congestion of lower lobes. *Heart*.—Large ante-mortem clot in right auricle, and a smaller one in left auricle. *Glands*.—Mesenteric glands enlarged and congested. *Intestines*.—Patches of congestion in lower part of small intestine. Large intestine much thickened and deeply congested throughout its course. A few superficial ulcers, especially near the ileo-cæcal valve.

Busey's case. Necropsy twenty-four hours after death. Aged two years, emaciated, abdominal walls retracted, and rigidity slight. *Brain*.—Weight 2 pounds $5\frac{1}{2}$ ounces, anæmic, effusion into arachnoid cavity (estimated) 1 pint, slight in ventricles. Black clots in all the sinuses, and a large white fibrinous thrombus at the junction of the right lateral with the petrosal sinus. *Heart*.—Weight $1\frac{1}{2}$ ounces; effusion into pericardium; white fibrinous clot in superior vena cava extending into right auricle and firmly attached to base of tricuspid valve. No blood in either ventricle, and valves intact. *Lungs*.—Weight $7\frac{1}{2}$ ounces, float in water; left normal, right contained in middle lobe a cheesy mass as large as a hen's egg; this lobe was firmly attached to pleura. No tubercular deposits. A cheesy bronchial gland as large as a pigeon's egg. *Abdomen*.—Abdominal walls thin and destitute of fat. Omentum contains but little fat. Mesenteric glands slightly enlarged and congested. Small intestines contain fæces, and nothing abnormal noted. Patches of intense inflammation all along the tract of large intestine from cæcum to anus. Liver anæmic, buff-colored; gall-bladder distended. Large deposits of pus at lower extremity of either kidney. Weight $1\frac{1}{2}$ ounces each.

FIG. 1.



Showing Dysenteric Ulcer of Colon.

MICROSCOPICAL APPEARANCES.—There is considerable loss of surface epithelium and of that lining the tubular glands. The glands frequently contain pus-cells and degenerated epithelium. The interglandular tissue is infiltrated

with serum and pus-cells. The mucous membrane softens, and necrosis extends for a considerable distance into it. Here the glands are broken down, their confines are lost and they may fall out or remain incarcerated in cast-off epithelium, mucus, and pus. There may be ulceration accompanying these changes. The ulcers are shallow and without well-defined borders. They result from softening, suppuration, and exfoliation of the tissues into the sub-mucosa or even down to the muscular coat. The solitary follicles are swollen to the size of two or three millimetres in diameter, and vary in color from transparent gray to opaque white. The swelling is due to an increase of round-cells or hyperplasia of lymphatic tissue. Large epithelial and pus-cells, mingled with lymphocytes, may be seen in the nodules. If the destructive process continue, the epithelium over the lymph-nodules breaks down and an ulcer is formed. The lymph-nodules then appear elevated, with a central depression.

Symptoms.—The onset may be sudden, without premonitory symptoms, accompanied by one or more chills or preceded by diarrhoea. The temperature is usually elevated two or three degrees, depending upon the intensity of the inflammation; the pulse soon becomes rapid, small, and compressible; the strength is rapidly diminished; and the face presents a pinched, pallid, and anxious expression. The tongue is moist and covered with a whitish fur. There is seldom abdominal pain or tenderness on pressure. There is constant desire to go to stool, with pain and straining during and after evacuation. The stools, which at first contain faecal matter, soon become small, frequent, odorless, and consist of blood, mucus, and pus. Sloughs are rarely seen. The stools vary in number from eight or ten to forty or fifty in the twenty-four hours. As the inflammatory process advances to ulceration the stools contain shreds, resembling "washed raw meat," mingled with blood and pus, and may be passed involuntarily. The straining now becomes more severe, and prolapse of the rectum frequently results from it. The abdomen becomes tympanitic, and tenderness marked along the entire course of the colon. The tongue becomes dry, with brown centre and red margin. Vomiting may supervene, and prove to be intractable. The pulse becomes rapid, thready, and intermittent, and syncope threatening. The respirations become sighing and the voice inaudible. The eyelids are partially closed, and the pupils are widely dilated. The child becomes restless, and tosses from one side of the bed to the other, and delirium or convulsions may be present. The urine is high-colored and scanty, or there may be total suppression, with vesical tenesmus.

If examined microscopically, the typical "dysenteric stool" contains traces of ingesta, various kinds of bacteria, fat, epithelial cells, round cells, mucus, blood-corpuscles, and pus-corpuscles mingled together.

CASES. Nellie E—, aged eighteen months, had been suffering several days with loose bowels. The evacuations becoming frequent, small, bloody, and slimy, the parents called in a physician. He found that she had a dozen or more dysenteric stools daily, accompanied by great tenesmus, and that there was marked prostration. The disease yielded to treatment, and she recovered in three days.

John B—, aged twenty-two months, had had frequent bloody discharges for several days, and had been dosed with numerous remedies for "summer complaint" which had been prescribed by other physicians for other people's children. As he rapidly grew worse, I was summoned. Found him running about the room, but he would frequently assume the squatting position and strain. He had had twenty bloody, slimy, offensive stools, and as many of "a stain of blood and slime," during the previous twenty-four hours. The pain did not seem to be so severe, but he would strain until drenched with perspiration. He could not be kept in bed. Finally, his symptoms became so much worse that he was held by one of his parents, but not in recumbency. This modified rest did but little good, as the rectum was soon prolapsed to about half an inch. After

exhausting the usual methods of treatment the disease succumbed to suppositories of cocaine and ergotin, on the eighth day of my service.

Lottie E—, aged four years, was seen forty hours after the dysenteric symptoms began. She was now having frequent, offensive, muco-sanguinolent stools, accompanied by exhausting tenesmus. The pulse was frequent and small, and the temperature was not 100° F. The symptoms rapidly grew worse and she seemed liable to die at any moment from cardiac failure. The rectum protruded, became œdematous, and blood exuded from the mucous membrane. The prolapsed gut seemed to be about two inches in length. On the ninth day of the disease the dysentery yielded to treatment, but the prolapse lasted for a week longer.

Dimple G—, aged seven years, had been sick for five days with dysentery. The bloody discharges had increased in number, the pain had become more intense, the desire to stool more imperative, and the evacuations were characterized as small, bloody, and slimy. She was suffering from strangury produced by turpentine stupes, which had been used for several days. She had had two hundred and eighty-one bloody, slimy stools in thirty-six hours (four hundred and sixty-three during the five days of her illness). Dr. D. obtained this history, and the following day called me in consultation. She now had the appearance of being extremely ill. Her pulse was small, frequent, and compressible; the eyes were sunken and the pupils dilated; the cheeks were pale and sunken, and the lips livid and pinched; the tongue was slightly coated and very dry, and thirst was intense; there was nausea, but not vomiting, although she had vomited in the early part of the illness; the abdominal walls were flabby, and there was no pain upon pressure over the abdomen. She had not slept for several days, and was continually begging for sleep. The discharges were involuntary and had become so frequent that cloths were kept under the nates to catch them; they were small, bloody, and offensive. There was great pain and straining. The voice was almost inaudible, and the respiration was sighing. Cerebral anemia was well marked. She had frequent attacks of syncope, although not permitted to raise her head from the pillow. Her condition was so critical that a physician remained in her room. Stimulants and food were systematically given until the stomach and rectum refused to retain them, when brandy and, finally, ether were given hypodermatically. The attacks of syncope became more and more frequent, and she died of exhaustion and heart failure seventy-two hours after the first consultation.

Diagnosis.—In sporadic cases of dysentery there may be some difficulty in differentiating it in its early stage from acute intestinal catarrh, but when the characteristic stools have once made their appearance all doubt will disappear. In dysentery the stools contain mucus, blood, pus, and small masses of fecal matter, and are odorless or have a "fresh-meat odor;" tenesmus is always present, a small quantity is expelled from the bowel after a violent effort, and the patient is bathed in a cold, clammy sweat, is exhausted, and probably faints. In acute intestinal catarrh the evacuations are larger; the blood, when present, is in streaks and not mixed with mucus; the pain is more intense and paroxysmal; and tenesmus is seldom present.

The differentiation of sporadic from epidemic dysentery can be made by the prevalence of the latter in the community.

Prognosis.—The prognosis in acute catarrhal dysentery in children is usually favorable. The ordinary duration is from eight to ten days, but it may prove fatal in twelve, twenty-four, forty-eight, or seventy-two hours. The favorable symptoms are absence of foul odor, diminution in frequency and improvement in the character of the stools, and disappearance of tormina and tenesmus; the absence of nervous depression and of anxious and careworn expression of countenance; and increase of heart-power and arterial tension.

The unfavorable symptoms are increased blood-loss, ashy aspect of countenance, nausea, vomiting, hiccough, tympanitic and tender abdomen, nervous depression, sleeplessness, tossing about the bed, moaning, delirium, convulsions or other marked cerebral disturbances, and suppression of urine. When convulsions appear, death is not far distant. Busey observes that in many cases death takes place under exactly similar circumstances—viz., one, two, or

three convulsions, followed by coma and death, and in none of his cases did consciousness return after the first convulsion.

II. AMŒBIC DYSENTERY.¹

This form, which is also known as tropical dysentery, is characterized by the presence in the stools of the *amœba coli* (Lösch), *amœba dysenteriae* (Councilman and Laffeur). It is this form which occurs in such fatal epidemics in the tropics. "The amœba is a unicellular, protoplasmic, motile organism, from ten to twenty micro-millimetres in diameter, consisting of a clear outer zone, ectosarc, and a granular inner zone, endosarc, containing a nucleus and one or more vacuoles. It was first described by Lambl in 1859, and subsequently by Lösch, who considered it the cause of the disease"—(Osler). The disease is not infrequently seen in Europe and North America, but its home is in tropical and subtropical countries. The most frequent source of infection is unquestionably the drinking-water.

Morbid Anatomy.—Like the other varieties, the lesions are situated in the colon, but in some cases they are also found in the lower portion of the ileum. These lesions consist in ulcers, which result from infiltration into the submucosa. At first small elevations appear along the mucosa; the mucous membrane covering them sloughs off, exposing an ulcer with a grayish-yellow floor. Councilman divides these ulcers into four forms: (1) "Ulcers characterized by cellular infiltration, softening, and cavity-formation in the submucosa; these have a small opening in the mucous membrane and often communicate with neighboring ulcers by passages in the submucosa. (2) Ulcers with slight undermining of the edges, representing simple excavations in the thickened submucous tissue. (3) Ulcers with smooth sides and clean bases. (4) Ulcers with extensive adhering sloughs." These simply represent different stages of the same process. The non-adjacent mucosa remains unaffected.

Osler says the "microscopical examination shows a notable absence of the products of purulent inflammation. In the infiltrated tissues polynuclear leucocytes are seldom found, and never constitute purulent collections. On the other hand, there is proliferation of the fixed connective-tissue cells. Amœbæ are found more or less abundantly in the tissues at the base of, and around, the ulcers, in the lymphatic spaces, and occasionally in the blood-vessels.

"The lesions in the liver are of two kinds: firstly, local necroses of the parenchyma, scattered throughout the liver and possibly due to the action of chemical products of the amœbæ; and, secondly, abscesses. These may be single or multiple. When single they are generally in the right lobe, either toward the convex surface near its diaphragmatic attachment or on the concave surface in proximity to the bowel. Multiple abscesses are small and generally superficial. In an early stage the abscesses are grayish-yellow, with sharply defined contours, and contain a spongy necrotic material, with more or less fluid in its interstices. The larger abscesses have ragged, necrotic walls, and contain a more or less viscid, greenish-yellow or reddish-yellow purulent material mixed with blood and shreds of liver-tissue. The older abscesses have fibrous walls of a dense, almost cartilaginous toughness. A section of the abscess-wall shows an inner necrotic zone, a middle zone in which there is great proliferation of the connective-tissue cells and compression and atrophy

¹ The writer has depended almost entirely upon the valuable contributions of L. Emmett Holt, Osler, and Laffeur and Councilman in preparing the sections on amœbic and diphtheritic dysentery.

of the liver-cells, and an outer zone of intense hyperæmia. There is the same absence of purulent inflammation as in the intestine, except in those cases in which a secondary infection with pyogenic organisms has taken place. The material from the abscess-cavity shows chiefly fatty and granular detritus, few cellular elements, and more or less numerous amoebæ. Amoebæ are also found in the abscess-walls, chiefly in the inner necrotic zone. Cultures are usually sterile. Lesions in the lungs are seen when an abscess of the liver—as so frequently happens—points toward the diaphragm and extends by continuity through it into the lower lobe of the right lung. The gross and microscopical appearances are similar to those of the liver.

Symptoms.—Sometimes the onset is sudden and at other times gradual. The severer forms are characterized by a sudden onset. The diarrhœa intermits, while loss of strength and emaciation are progressive. Moderate fever is usually present, although some cases are unattended by this symptom. In some, tormina and tenesmus and nausea and vomiting are marked at the onset, while in others they are not observed. Twelve or fourteen grayish-yellow stools, containing blood and mucus are voided daily. This condition persists for weeks. The amoebæ are found in great numbers in the stools during the diarrhœal attacks, but gradually decrease, and finally disappear as the attack subsides.

Diagnosis.—This form is differentiated from the catarrhal by the frequent exaggeration and remission of the diarrhœal symptoms, but more especially by the presence of amoebæ in the stools.

Prognosis.—The duration varies from six to twelve weeks. The prognosis is not as favorable as in the catarrhal form; and convalescence is slow, owing to the depletion, the relapses, and the chronic tendency.

III.—DIPHThERIC DYSENTERY.

Diphtheritic or croupous inflammation of the intestinal tract is the most fatal variety. It usually begins in the intestine, but may result from diphtheria situated in the mouth, pharynx, or nose.

Morbid Anatomy.—Macroscopically, there is nothing significant in the appearance of the intestinal contents unless patches of pseudo-membrane are found upon washing. The stools vary in color from yellowish-green to greenish-brown, and consist of mucus, fecal matter, occasionally digested blood—seldom pure blood—and perhaps pieces of pseudo-membrane.

The lesions are situated over the entire colon and the lower portion of the ileum, but are most numerous near the cæcum. The intestinal wall is greatly thickened and the rugæ are obliterated. Small grayish-white, opaque masses are seen upon the congested mucosa. These masses cling to the surface, and can only be removed by tearing off a portion of the mucous membrane. These small areas may coalesce and form a patch which involves the greater part of the intestine, converting it into a thick, inflexible tube. Where the membrane is extensive it is marked by numerous transverse and longitudinal fissures, which give it the appearance of separate patches. The mucous membrane devoid of the patch is intensely congested and roughened, or the only changes may be confined to the diphtheritic areas.

MICROSCOPICAL APPEARANCES.—There is infiltration of the mucosa, and in some cases, of the submucosa. The pseudo-membrane is composed of fibrin, necrotic cells, and sometimes blood-corpuscles. The tubular glands are usually unrecognizable, but their remains may often be detected in the necrotic masses. The thickening of the intestine is due to the infiltration of the submucosa, the

dense mass of fibrin, the engorged blood-vessels, and extravasations of red blood-corpuscles. Ulcers are seldom present in children, but when found are usually of the follicular variety.

Symptoms.—This form is not seen in infants and is uncommon in children. In some cases the onset is insidious, and may be mistaken for the catarrhal, while in others it is abrupt and alarming. The symptoms are similar to, but more severe than, those of the catarrhal or amœbic. The pathognomonic symptom is the presence of pseudo-membrane in the stools.

TREATMENT OF DYSENTERY.

PROPHYLAXIS.—Acute catarrhal dysentery may often be avoided by promptly and energetically treating the simpler forms of intestinal disease. It too often happens that disorders of digestion are regarded as trifling, and skilled assistance is only summoned when the signs of severe anatomical lesions become manifest.

Hygiene.—Personal and domiciliary hygiene should be carefully supervised. The child should be bathed at least once a day, and in very hot weather twice. His clothing should be changed sufficiently often to protect him from sudden variations in temperature; especially is this true during the cool nights of autumn. If not already too ill, he should be removed from the heat of the city to some salubrious resort in the mountains or at the seashore. If circumstances compel him to remain at home, he should be placed in a room where pure, fresh air will be admitted freely. An occasional sponge-bath of equal parts of alcohol or bay-rum and water will prove to be grateful, and will reduce the body heat as well as allay nervous irritability. The infant's diapers should be removed and placed in a disinfecting solution as soon as soiled, and in older children the evacuations should be immediately disinfected. For this purpose solutions of carbolic acid, 1:20, corrosive sublimate, 1:500, milk of lime, or some other germicidal drug must be kept in some convenient place.

It is none the less important that the hygiene of the premises should be scrupulously watched and every means possible used to prevent the accumulation of filth.

Rest.—Rest in the recumbent posture must be enjoined from the start. The stools should be passed in this posture, as any other will increase the pain and straining.

DIETETIC.—The diet should be prescribed in the very beginning, and but little discretion given to parent or nurse. The nursing infant should continue at the breast unless some condition of the mother, or of her milk, contraindicates it. In all others sterilized, Pasteurized, or peptonized milk, beef-tea, beef-juice, or mutton-broth, or all alternately, should be given in small quantities at frequent intervals. Care should be taken not to overfeed, lest harm be done. When the blood and mucus have disappeared from the stools, we may gradually but cautiously return to a more liberal and mixed diet. A liberal supply of natural mineral water, distilled water, or boiled city water must be allowed. If the child refuse or is unable to swallow, food must be administered by *gavage*—a method not at all difficult, and attended with satisfactory results.

MEDICINAL.—There are no specifics for this disease, although different remedies have been specially recommended by different writers. Some believe the best results are to be obtained from ipecacuanha, others from opium, purgatives, or vegetable or mineral astringents, while, latterly, many rely upon the administration of intestinal antiseptics, as salol, mercuric chloride, naph-

thol, and sulphocarbolate of zinc. While it may be admitted that all of these methods have their advantages in individual cases, still, no one has proved to be uniformly successful in the hands of those who treat the greatest number of cases.

Usually the first indication for treatment is the removal of undigested or indigestible food from the alimentary tract. For this purpose the mild saline purgatives are especially indicated, or a stronger purgative, as for a child aged 6 years :

R_y. Pulv. ipecac. gr. ss.
 Mass. hydrarg. gr. iij.
 Pulv. aromatic. comp. gr. v.
 Sacchar. alb. gr. xv.—M.
 Ft. chart. No. X.

Sig. One every two hours.

R_y. Tinct. opii deodorat. gtt. xij.
 Olei ricini f̄ss.
 Pulv. acaciæ q. s.
 Aquæ rosæ q. s. ad f̄ij.—M.

Sig. Tablespoonful every two hours.

As soon as the scybala and undigested masses have been removed this treatment should be suspended.

Of the mineral astringents the subnitrate of bismuth, in large doses, holds the highest rank. The author has, at times, received benefit from the following, which is both astringent and antiseptic :

R_y. Plumbi acetatis gr. iv.
 Acidi acetic. q. s.
 Acidi carbolic. gr. ij.
 Liquor. calcis q. s. ad f̄ij.

Mix the first, third, and fourth, and add enough of the second to make a perfectly clear solution.

Sig. Teaspoonful every three hours.

If the pain and straining are intense, relief may be derived from the following :

R_y. Cocain. muriat. gr. j.
 Ext. ergot. aq. gr. x.
 Ext. opii aq. gr. ij.
 Aristol gr. v.
 Olei theobrom. q. s.—M.
 Ft. Suppos. No. X.

Sig. One every two or three hours.

Stimulants are imperative, but should be administered with great care. The dose of whiskey or brandy must be regulated by the age of the child and the exigencies of the case. When these fail, the more powerful and diffusible cardiac stimulants should be given, perhaps hypodermatically.

LOCAL.—In the light of modern science the most rational treatment of dysentery is intestinal irrigation. By it the irritating contents of the colon and rectum are washed out and the pain and straining are mitigated, and in

some cases entirely relieved. A distinction has been drawn by Dr. W. W. Johnston, of Washington, D. C., between intestinal irrigation and injection. The former is more correctly the application of a running stream to the inflamed gut, in which the fluid has free egress, while in the latter the fluid is introduced to painful distention. In the former a second tube permits a free outpouring and in the latter the fluid must escape between the nozzle of the syringe and the anal sphincter or be forcibly expelled by the disabled intestine after the tube is withdrawn. The former is preferable when the lesions are below the sigmoid flexure, and the latter when they are above it.

To irrigate the rectum a double injection-tube, attached to a fountain syringe, should be passed from three to five inches into the bowel, through which a current of water is kept flowing at the pleasure of the operator. As the passing of such an instrument is nearly always attended with great pain, it is better to use two soft rubber catheters, well oiled; the larger is attached to the tube of the syringe, while the smaller is used as the escape-pipe. Pressure on the flexible tubes by the operator's fingers will regulate the inflow and outflow of the fluid.

To irrigate the entire colon in a child of eight or ten years it is necessary to inject one or two pints immediately after a stool, but an infant requires much less. The author has never succeeded in injecting such large quantities into the bowel, but has obtained very satisfactory results from small quantities by forcing it to be retained for a short time, by pressing a napkin against the anus. This fluid must be slowly injected, so as to allow the inflamed and infiltrated coats to adapt themselves to the increased tension.

The irrigating apparatus being ready, the child is placed on his left side, with the hips on a plane higher than the body, or, still better, in the knee-chest posture, so as to favor the inflow. The first irrigation should be given by the physician, who will thus instruct the nurse to follow his particular method. When the pain and tenesmus are severe, and the introduction of the tube intensifies both, the rectum may be partially or completely anæsthetized by suppositories of ice, aristol, euphrophen, or cocaine, or by the injection of a 2 or 4 per cent. solution of cocaine or carbolic acid.

The frequency of irrigation is best determined by the number of stools, the object being to prevent the patient from having stools by washing out the intestinal contents through the tube. At first the irrigation should be given after every stool; then, as the pain and tenesmus lessen and the blood and mucus decrease, it must be given at longer intervals; and, finally, when the movements border on the natural, a daily irrigation for a few days may prevent a relapse.

Hot or cold water, either plain or holding in solution one of the numerous antiseptics, may be used as the irrigating fluid. In some cases very hot water will afford marked benefit, while a large number, in the author's experience, have received almost immediate relief from cold or ice-water. The temperature of the water must be gradually lowered when irrigating the infant's bowel, as the shock from ice-water might prove fatal.

Every writer has a favorite antiseptic for dysentery, but mercuric chloride, 1:10,000, is most extensively employed. The bowel must be quickly and thoroughly emptied of this fluid to insure protection against its poisonous effects from absorption. Some of the other antiseptics are carbolic acid, boracic acid, hydrochloric acid, salicylic acid, aseptol, thymol, sulphocarbolate of zinc, nitrate of silver, alum, quinine, and creolin. While it is advisable to use some antiseptic solution in the graver forms, the great benefit to be derived from irrigation in catarrhal dysentery is the cleansing.

In amoebic dysentery, Councilman and Lafleur have used solutions of quinine, 1:5000, 1:2500, 1:1000, in five cases. In 3 cases improvement was marked, in 1 the injections were suspended owing to a fatal complication, and in the other the amoebæ did not decrease during the quinine injections. Lösch found by experimentation that solutions of quinine, 1:5000, would kill amoebæ outside of the body, so Councilman and Lafleur were led to use it by intestinal irrigation. The patient should be placed in the knee-chest posture, and a half-pint or a pint of the quinine solution injected thrice daily, the enema being retained for fifteen minutes. These writers claim that the enemata kill the amoebæ in the intestine, but have little or no effect upon those in the tissues.

In diphtheritic dysentery the same rules of treatment that are recommended in the other forms are applicable, but must be more vigorously employed. Irrigation with solutions of mercuric chloride, silver nitrate, or hydrogen peroxide seems to be the most rational procedure.

CHRONIC CONSTIPATION.

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CHRONIC CONSTIPATION, or the absence of a regular, periodical expulsion of faecal excrement from the bowels, is very prevalent in infancy and early childhood. In childhood a daily evacuation should be the rule, whilst in infancy two, three, and sometimes even four, motions are usual.

Excluding acute bronchitis, habitual constipation is the most common ailment met with in early life. It is rather an aberration from the normal functional activity of the bowels than an essential disease, but if not relieved may in time seriously affect the general health of the patient by interfering with the functions of other organs and with the processes of nutrition, and, as an ultimate result, life even may be endangered. It often proves a very intractable disorder, and, despite all that is done, it may continue throughout the period of childhood, interfering with healthy development as well as with comfort. Children who are artificially fed are more prone to constipation than those who are suckled at the breast.

Etiology.—The cause of the constipation may exist in the person of the patient, or the condition may be the result of some extraneous influence. We will first speak of the former.

In the child the small intestine is comparatively longer and its lumen narrower than in the adult, and its walls are feeble and not so thick. Again, the ascending and the transverse colon are shorter, while the descending colon is longer relatively than in the adult. Finally, the many curves of the intestinal canal, the deep cul-de-sac in the sigmoid flexure just above the rectum, and the contracted conformation of the pelvis in children, with the consequent crowding of the intestines into a relatively small space, are well known. All of these anatomical peculiarities act as causal factors. Another element of importance in infancy, but which lessens in force as the child develops, is feeble peristalsis, due to the imperfectly-developed state of the muscular coat of the intestines.

A diminution in the amount of intestinal secretions, especially of the bile, favors the occurrence of constipation, for under such conditions the faecal mass becomes hard and scybalous, and is apt to be too long retained. Then, too, if fermentable food be taken, large quantities of flatus are generated and pain and abdominal distention attend the constipation.

Certain pathological conditions, obstructive in nature, are often present. Among such conditions are tumors, congenital malformations, and uterine retroflexions; constricting bands resulting from acute or chronic peritonitis; intestinal displacements, stenosis at the ileo-cæcal valve, and a nest of lumbricoid worms. Local disorders seated in the rectum may also cause constipation. Chief among these is fissure of the anus, for in this disease, as pain is produced when defecation is attempted, the patient refrains from the act of evacuation and the constipated habit is gradually formed.

In diseases of the central nervous system, as tubercular meningitis, hydrocephalus, microcephalus, and myelitis, which interfere with the innervation of the abdominal and intestinal structures or which produce a spastic contraction of these parts, constipation is generally present. The various constitutional dyscrasias, as tuberculosis, rachitis, syphilis, and the like, may, by weakening the muscles engaged in the act of defecation, act as causes.

Any condition depriving the organism of water in large quantities renders the fæces dry and predisposes to sluggish bowels: profuse perspiration and the polyuria of diabetes come under this head. Want of attention in infancy and the neglect to respond to the calls of nature on the part of older children are potent factors, for by repeated stimulation and over-distention of the rectum by its contents, its muscular activity is worn out and an atonic condition is the result. Constipation sometimes results from diarrhoea. In such cases it is due to atony and paresis of the muscular envelope of the intestines caused by excessive and persistent irritation. Insufficient peristalsis, accumulation of fæces, dilatation of the entire bowel or of certain parts, accompanied by reflex symptoms due to interference with other functions of the body, are additional factors conducing to this result. Some authors say that all cases of habitual constipation are accompanied by a considerable amount of chronic irritation and subacute inflammation of the cæcum and colon and neighboring cellular tissue. The effect of this is to reflexly arrest peristalsis.

We will now consider what may be called the *extrinsic causes* of constipation, or those which operate from without the body. Constipation in infants at the breast may be the consequence of a constipated habit on the part of the mother. In such cases the maternal milk may be deficient in fat, sugar, or salt. In older children improper food is a very frequent cause. On the one hand, food may be given to the child which after digestion leaves very little residue in the bowel, so that no stimulation of the intestines is produced for the expulsion of its contents. On the other, too coarse foods may be given, and the residue may be so great that by constant over-stimulation of the muscular coats of the intestines their tonicity is exhausted. Excess of farinaceous foods will act in this manner, and all foods that are prone to fermentation by producing accumulations of gas will hinder free action of the bowels. Lack of moisture in the intestinal contents, resulting from scanty ingestion of water, is another factor; and still more potent are the indiscriminate use of medicinal agents, especially castor oil and spiced syrup of rhubarb—laxatives having a secondary astringent action—and the repeated use of enemata, which destroy the natural sensibility and reflex activity of the rectum.

In older children an in-door, sedentary life, negligence in regard to the formation of a regular habit of evacuating the bowel, and a faulty posture at stool are active in producing the constipated habit. In regard to the last element, it may be said that in the physiological act of defecation the individual should assume such an attitude that every muscle of the back and abdomen which causes the bowel to be quickly and thoroughly emptied of its contents may be brought into action.

Pathology.—The pathological condition to be found in constipation varies from a simple hyperæmia to a catarrhal or even ulcerative condition of the mucous membrane of the intestines. Yet in many cases nothing whatever is to be discovered in the intestinal canal. The intestinal walls are apt to become thin, and some authors maintain that fatty degeneration of the muscular coat of the intestines supervenes, resulting in a loss of contractility and expulsive power. A swollen and distended condition of the bowels and a chronic inflammation, with induration and thickening in the region of the cæcum, are occa-

sionally met with. Herniæ, particularly umbilical, prolapsus ani, varicocele, fissure, cystitis, and hæmorrhoids may be the results of the violent and oft-repeated straining, and the liver may be pressed upward and congested from interference with the portal circulation.

Symptoms.—When constipation is due to obstruction, fæcal matter accumulates above the occluded point and produces distention of the abdomen, accompanied by eructations of gas, vomiting, impaired appetite, and a consequent deterioration of the general health. If the intestinal contents be composed of hard masses or contain coarse, undigested material, there is danger of complete obstruction which will place the patient in a very perilous position.

In mild cases of functional constipation there is simply a retention of the fæces in the rectum or lower bowel; then there are no marked symptoms with the exception of a sensation of fulness, distention, and weight in these parts.

Generally speaking, the symptoms vary in degree according to the gradation from the mild to the very grave forms of the disorder, but it is surprising how frequently even severe cases of constipation are unattended by serious symptoms. Very often, in consequence of local irritation from the retained fæces, a conservative purging is excited, and the patient suffers alternately from diarrhœa and constipation. The bowel, however, is not always fully emptied of its contents when such a diarrhœa occurs, and the retained fæces in time undergo decomposition, with the generation of noxious gases, which in turn distend and irritate the bowels and cause severe colic. Fæcal and gaseous distention also interferes with the action of the diaphragm, and produces labored respiration or even great dyspnœa; it may also obstruct the venous circulation in the viscera and interfere with the cardiac action and the circulation in the thoracic cavity, leading to palpitation of the heart, irregular pulse, and vertigo. Again, pressure upon the abdominal and portal venous systems hinders the return circulation from the lower extremities, and produces slight œdema of the ankles and feet; finally, obstruction of the portal ducts and vessels, with attendant resorption of bile, may give rise to jaundice. In aggravated cases of chronic constipation the pressure of the retained fæces may cause inflammation of the mucous lining of the gut, when abdominal tenderness and fever will be noted. Sometimes the inflammation extends to ulceration, or even perforation, with their attendant symptoms.

When a constipated patient attempts to evacuate his bowels, he will experience great tenesmus, and the expelled mass may be streaked with blood and smeared with mucus, indicating that the lining membrane of the rectum has suffered in the violent effort at expulsion.

In infants constipation is accompanied by fretfulness; the little patient draws up his legs in pain, and, if he be nervously irritable, is very prone to an attack of eclampsia.

In all cases of long-standing retention the fluid elements of the fæces are reabsorbed, to be eliminated from the body by other emunctories. When this occurs the blood becomes contaminated, and there is impairment of the general health, with the production of such symptoms as languor, a foul breath and furred tongue, headache, nausea, and more or less complete anorexia; irritability of temper or hypochondriasis and moroseness. The abdominal nervous plexuses also are affected, and the sufferer, when old enough, complains of fornication, fatigue, and pain in the abdomen and lower extremities.

Diagnosis.—While the recognition of the existence of constipation is of course very easy, it is often a difficult problem to detect the condition—the actual disease—leading to the functional disorder of the bowels; and this

problem must be correctly solved before successful treatment can be inaugurated.

Such conditions as hernia, hæmorrhoids, and continued tenesmus should always lead one to expect the presence of the constipated habit. On the other hand, all children who have small or infrequent fæcal evacuations are not constipated, as such features may be noticed when the food is too concentrated or is allowed in insufficient quantity.

Prognosis.—Simple idiopathic chronic constipation never endangers life. If, however, the condition depends upon some structural abnormality, the prognosis is more grave. In its consequences, both immediate and remote, constipation is of serious import. It will lead, as has been said, to fissures, hæmorrhoids, and other local troubles; it impairs the general health, and if not attended to early a lifelong habit is formed.

Treatment.—To secure, if possible, the removal of its cause should be our first consideration in the treatment of constipation. If, on account of structural or pathological reasons, this be impossible, our efforts must be directed to the minimization of its ill effects. In nurslings drugs should, as far as possible, be avoided, attention being paid to the food and to the diet of the mother or nurse. If the mother's milk be deficient in fat, sugar, or salts, her diet should be so modified that a larger quantity of these principles are presented for assimilation; she should also partake of laxative foods. If the child be nourished by a wet-nurse, the same ends may be accomplished by a change to one who is in an earlier stage of lactation and whose milk contains more fat and less albumin. When, in spite of these measures, the constipation continues, some simple laxative is indicated. I frequently use a little molasses or melted sugar and butter or sweet oil in teaspoonful doses. If the constipation be due to an insufficiency of fluids, as indicated by dry and brittle motions, it is wise to insist upon the child's being given water several times daily—an item often overlooked by parents and nurses. Sometimes oatmeal-water may be substituted for plain water with advantage, particularly in older children taking a mixed diet.

When these simple measures fail, the next resort is to suppositories or enemata, which act by local stimulation of the rectal muscles. Suppositories should be conical in shape and made either of soap or molasses candy, or should contain either gluten or glycerin. I prefer those containing glycerin, as being most prompt and efficient in action. But whatever variety is selected, it should be well oiled before insertion, and then gently introduced and pushed up well beyond the internal sphincter. Glycerin may also be used by injection, in the proportion of ten to twenty drops to two fluidrachms of water. Such an injection is followed in from five to ten minutes by a full and painless motion. The efficiency of the glycerin is due to its hygroscopic action; it abstracts water from the mucous membrane of the rectum, inducing hyperæmia of the part and increasing peristaltic action through nervous excitation. Cold-water injections are also recommended. These may be given at first three times, then twice, and finally once, daily until a cure is effected. The addition of a little table salt increases the activity of these enemas. As to the bulk of the injection, one or two fluidrachms will usually suffice in infants. Too large enemata not only dilate the bowel and paralyze its muscular coats, but may also give rise to much pain, and even interfere with the respiration and circulation.

If it be necessary to resort to drugs, the most simple are to be chosen, as small doses of calomel, castor oil, solution of citrate of magnesium, carbonate of magnesium, and phosphate of sodium, in properly graded doses. The last

remedy has given me great satisfaction in doses of one to five grains according to the age of the patient. I frequently administer it in the following combination:

R̄. Sodii phosphatis gr. xxiv.
 Syr. mannae f ʒiiss.
 Aq. anisi q.s. ad f ʒiij.—M.

Sig. One teaspoonful three times daily, for a child under one year old.

Calomel may be given in one-sixth grain doses several times daily, but must not be employed habitually; laxatives that can be used more freely are carbonate of magnesium in one- or two-grain doses in a little milk or aromatic water, and the solution of the citrate of magnesium in doses of one to four fluid drachms.

Older children must be trained to the formation of the habit of regular daily evacuation of the bowels at a fixed time. Neglect of this very important rule is very often the cause of constipation persisting through adult life, with its disagreeable train of symptoms. Attention should also be directed to the posture assumed in the act of defecation, in order that all the necessary muscles may be brought into play. It is important, too, at this age to encourage outdoor exercise, and to so regulate the diet that the child will receive plenty of water and an abundance of laxative food. In this class belong fruit, either in its natural state or cooked, oatmeal or cracked-wheat porridge, corn and brown bread, green vegetables, molasses, etc. Farinaceous foods must be restricted, but milk may be taken freely if the digestion be good. In the proscribed list come cheese, uncooked dried fruits, fruits having numbers of small seeds, and spices.

In the administration of medicine select the particular one that agrees best with the patient; seek the appropriate dose to secure an evacuation; then gradually reduce the dose until the constipation is ended. One of the most useful drugs is calomel, given alone or in combination with powdered rhubarb, half a grain of the former to one grain of the latter. This may be repeated several times daily, but care must be taken not to administer calomel repeatedly in either tuberculous or rachitic children. If any rectal irritation be present, compound licorice powder combined with sulphur is very useful. If flatus be present, carbonate of magnesium combined with asafoetida will afford relief. The fluid extract of cascara sagrada in one- or two-drop doses is a very good remedy. Dr. Earle of Chicago recommends "in the case of a child two years of age to clean out the bowels with two or three grains of calomel combined with a little compound licorice powder, followed for a few days with carbonate of magnesium ʒij in f ʒj of water, one to three teaspoonfuls daily until the bowels are relaxed. Then give non-astringent iron preparations, nux vomica, and possibly magnesium sulphate or cascara, until the cure is complete."

It has also been suggested that small doses of ipecacuanha, either alone or combined with calomel, are very useful.

When there is great distention of the bowels it will be of advantage to bandage the abdomen in order to assist in the restoration of muscle-tone. The colon may be punctured with a hypodermatic needle when its distention is so great that collapse is imminent from heart displacement. When there are large collections of fecal matter in the colon, the more active cathartics must be exhibited, accompanied by irrigation of the bowel through a rectal tube. If the feces are very hard, it is advisable to add to the fluid injected inspissated ox-gall in the proportion of ʒij to the pint. I frequently add to the ox-gall

the following mixture, which stimulates the bowel to relieve itself of its contents, and also helps to carry off flatus:

R̄. Ol. terebinthinæ fʒj.
 Tr. asafœtidæ,
 Ol. ricini āā fʒiv.—M.

Sig. Add to a quart of warm water, and use for irrigation.

The suds of ordinary brown washing soap may also be added to this mixture. If the rectum be impacted, instrumental and manual assistance must be given; injections of small quantities of yeast have been also used with success. Gradual dilatation of the sphincter has also been successfully employed. If constipation be accompanied by the symptoms of indigestion, the diet should be revised; pepsin with muriatic acid and cascara or taraxacum should be prescribed. I again desire to call attention to the phosphate of sodium; in older children it may be given in doses of from five to eight grains dissolved in water.

The constipation which succeeds a diarrhœa requires the use of tonics. Of these, strychnine stands first in efficacy, administered either alone or in the favorite combination of iron, quinine, and strychnine. When atony of the muscular coat occasions the trouble, nux vomica combined with belladonna, ergot, and phosphorus are very valuable remedies.

As each case must be treated on its own merits, many of the cathartic remedies which have not been alluded to by name will undoubtedly meet special indications in special cases. Thus when there is an interference with the hepatic functions the following is an excellent prescription:

R̄. Resinæ podophylli gr. j.
 Alcohol fʒiiss.
 Syr. rubi idæi q. s. ad fʒiij.—M.

Sig. A teaspoonful to a dessertspoonful every morning, according to the obstinacy of the constipation.

When a copious evacuation is desirable the following is recommended:

R̄. Tr. nucis vomicæ ℥xij.
 Tr. belladonnæ ℥xxiv.
 Inf. sennæ fʒj.
 Inf. calumbæ q. s. ad fʒiij.—M.

Sig. One teaspoonful for a dose.

The constipation which attends the various diathetic conditions demands individual attention, but by no means to the exclusion of the diathesis itself. Cod-liver oil and the syrup of the iodide of iron, both somewhat laxative in nature, are especially useful in these cases. A very good formula is that prescribed by Dr. J. Lewis Smith:

R̄. Olei morrhuæ fʒij.
 Liquor. calcis,
 Syr. calcii lactophosphatis āā fʒj.—M.

Sig. Give from one-quarter to one teaspoonful three times daily, according to age of child.

For anæmic children mineral waters containing iron are beneficial. Thus Friedrichshall is serviceable, as it has a tonic and laxative effect, and also

favors the elimination of uric acid. In such cases a mixture of sulphate of magnesium, sulphate of iron, and tincture of nux vomica is also serviceable.

Galvanism has its use in the treatment of constipation. The negative pole is passed well up into the rectum, and the positive along the course of the colon over the abdomen, for the negative pole excites local contraction, and the positive pole peristalsis. Galvanism is to be preferred to faradism, being more efficacious.

In conclusion, attention must be directed to one of the most important measures used in the treatment of chronic constipation—namely, massage of the abdomen and its contents. The technique of massage in children, though it differs in no essential particular from the same procedure in adults, should be modified in conformity with the position of the digestive organs at the various periods of the child's life. As the main cause of constipation in children, exclusive of the weak muscular coat of the bowel, resides in the descending colon, it is rarely necessary to practise the manipulations on the right side of the abdomen. The application of massage for as short a time as three minutes has been known to produce the desired effect, and the sitting should not last more than ten minutes. It may be repeated two or three times a day. The method of application is as follows: The operation is preferable before nursing or feeding, excepting when the child is very fretful or when the abdominal walls become very tense on handling. In such cases it can be accomplished during the act of feeding, for when the walls are very tense nothing can be effected. The hands should be clean, warm, and dry. The resistance and rigidity of the muscles will determine the amount of pressure to be used. The production of pain should be avoided; hence the pressure should be gradually made, and until the child becomes accustomed to it the manipulation should be very gentle. The fingertips placed upon the skin of the abdomen are moved about with the skin over the intestines, but *not rubbed*. For the first two or three minutes concentric circles are described by the manipulation in the region of the umbilicus; then in a similar manner the descending colon is treated, more pressure being made in the downward than in the upward movement. More manipulation is required in the left iliac fossa than elsewhere, for obvious reasons. The cæcum and ascending colon may at times also require to be manipulated in the same way. In older children sudden tapping of the abdominal walls with the fingertips, which will excite an instantaneous contraction of the abdominal muscles, has been found to be of value. The results obtained by massage have been very gratifying, and it should always be added to whatever other treatment may be instituted at any period of infancy or childhood.

SIMPLE ATROPHY.

By LOUIS STARR, M. D.,

PHILADELPHIA.

SIMPLE ATROPHY, or the slow wasting commonly termed “marasmus,” is a familiar occurrence in hand-fed babies, and one of the most frequent causes of death in early infancy. It is a condition in which there is extreme wasting of the soft tissues of the body, either without special organic lesions or with catarrhal inflammation of the mucous membrane of the gastro-intestinal canal.

Etiology.—Wasting usually occurs during the first twelve months of life, though it may begin in the second year, and is most frequently encountered among children of the poor. It arises both in breast-fed babies and in those brought up by hand, being in either case due to insufficient nourishment. The child wastes because he is starved.

Food can be insufficient in two ways: first, when it is supplied in amounts too limited to meet the demands of the system; and second, when it contains a minimum of the elements essential to nutrition or presents them in a form ill adapted to the feeble digestive powers of infancy. For example, nursing infants waste in consequence of feeding either from a breast that yields too little good milk, or from one that secretes abundantly a poor, watery fluid entirely unfit for nourishment. With artificially-fed children, on the other hand, it rarely happens that the quantity of food is too small; the fault lies, rather, in the direction of quality. Undiluted cows' milk, milk thickened with starchy materials, farinaceous foods, and even table food—meat, vegetables, and bread—are given to babies a few weeks or months old. Now, all of these are highly nutritious, but the digestive apparatus is not sufficiently developed to prepare them for absorption. They are strong foods, adapted to nourish and strengthen much older children and adults, but as the infant cannot appropriate them, he starves no less surely, if more slowly, than when taking no food at all. Such aliment also, while remaining undigested in the stomach and intestines, undergoes fermentation, with the formation of irritant products, causing vomiting or diarrhoea—conditions that still further lower the vital powers and hasten atrophy.

It is often possible to trace the disease directly to want of cleanliness in the feeding apparatus, and especially to the use of a form of bottle that has until lately been very popular in this country, as it is still in England. This bottle has, in place of a plain gum tip, an arrangement of glass and rubber tubing of small calibre. One extremity of the rubber tubing, which is eight or nine inches long, terminates in a small nipple-shaped tip and bone shield; the other, after penetrating an ornamental rubber cork, is fitted to a bit of glass tubing long enough to extend quite to the bottom of the bottle. By this plan the trouble of holding the bottle and keeping it at a proper angle during feeding is avoided. This seeming advantage, though, is counterbalanced both by the minor drawback that the child, left to itself, is apt to continue suction long

after the bottle is exhausted, thus swallowing a quantity of air, and by the greater disadvantage that the tubing can never be kept clean.

For a number of years the author made it a rule to ask for the bottle of every hand-fed infant presented for treatment, and few days passed without his seeing several of the complicated contrivances referred to. In almost every instance, notwithstanding the most careful and frequent cleansing, a sour odor could be detected, and if milk were present it contained numerous small curds; while in cases of carelessness the odor was intolerable, and the interior of the tubing was encrusted with a layer of altered curd. With simple bottles and tips, on the contrary, alterations in the character of the milk and coating of the interior of the tips were very infrequent. As there is little difficulty in keeping the bottles themselves clean, there can be only one reason for this difference—namely, in the simple instrument the nipple is readily removed and as easily inverted and cleaned, but in the other there is no way of cleaning thoroughly the twelve or more inches of fine tubing. The latter cannot be inverted, and the passage of a stream of water or of a stiff brush only imperfectly removes the milk clinging to the interior. This, of course, soon undergoes decomposition, and in this state quickly inaugurates change in the next supply of milk placed in the bottle. It is evident that a constant supply of food, no matter how good originally, thus rendered acid and partially curdled, must, like an excess of farinaceous or other unsuitable food, produce irritation of the alimentary canal, interfere with the processes of nutrition, and lead to a state in which the features of wasting and disordered digestion are combined.

The custom of preparing in the morning, without sterilizing, a supply of food sufficient for the whole day is another fruitful cause of atrophy. If this be done, no matter how carefully the mixture be proportioned or how well adapted to the age and digestion of the child, it becomes unfit for consumption after standing eight or ten hours. The change may or may not be appreciable to the senses, but test-paper will always show acidity and the microscope demonstrate the existence of actively-moving bacteria. Again, food upon which a child has thrived for three or four months, perhaps, can become unsuitable, and consequently lead to wasting, if the digestive powers be suddenly reduced by an intercurrent disease.

Wasting, while it is less serious in babies suckled at the breast, frequently occurs in a modified form under these circumstances. There are several additional causal factors. Thus, an infant may be given to a wet-nurse whose own baby is much older than her foster-child. In this case the milk is too strong, for it is a well-known fact that as lactation advances human milk becomes proportionately richer in curd and cream, and the nursling, unable to digest and assimilate it, ceases to thrive, and may even, in consequence, suffer from indigestion or diarrhœa. Human milk is also affected by dietetic and emotional influences, and, altering with the state of the general health of the mother, may deteriorate in quality or otherwise become unfit for food. Finally, it happens at times that, although the mother may be healthy and have an abundant breast, and although the infant may be robust, yet it does not thrive on the milk supplied. Here the fault is generally an over-richness in cream. While noting these facts, it must be remembered that in many cases of wasting in nursing infants the fault is not with the mother's milk, but in the digestive organs of the child, an attack of catarrh having temporarily impaired the process of digestion. Without care and proper management the derangement may be prolonged, and not infrequently leads to unnecessary weaning.

Morbid Anatomy.—After death the muscular and other tissues are found

in a state of atrophy, and there is a total disappearance of normal fat from the body. Fatty degeneration of the kidneys, lungs, and brain may be discovered; the stomach is sometimes ulcerated, and hæmorrhagic effusions into the cranium are not uncommon.

Symptoms.—The clinical features differ materially according to whether the element of insufficiency be one of quantity or quality. They may, therefore, be divided into two classes—viz. those developed by food that is suitable but not sufficient, and those resulting from unsuitable food.

The first group of symptoms is most frequently encountered in children who have been nursed at the breasts of feeble or overworked mothers, in whom the milk is often both scanty and of poor quality. There is a gradual loss of plumpness, the muscles grow flaccid, and there seems to be an arrest of growth. The face is white, the lips pale and thin, the skin harsh and dry or too moist, and the anterior fontanelle level or slightly depressed. The temper is irritable and sleep restless and disturbed; or the child is abnormally quiet, dozing constantly, and sucking his fingers until they become raw. When nursed the child seizes the nipple ravenously; then, if there be little milk, he quickly drops it to cry passionately, as if disappointed at not being able to satisfy his hunger; but if the milk be abundant, though thin, he will lie a long time quietly at the breast, and often fall asleep with the nipple in his mouth. The bowels are inclined to constipation, the stools being scanty, hard, and dry. Physical signs connected with the chest and abdomen are negative, and no indication of disease of any special organ of the body can be detected.

In the second class, features of wasting are associated with those of irritation of the alimentary canal, and the symptoms altogether are much more grave than in cases of the preceding group. The subjects are almost invariably hand-fed infants. Emaciation progresses with a rapidity and to an extent depending upon the original strength of the child's constitution, the age at which artificial feeding was begun, and the sort of food employed. It is often so extreme that an infant several months old weighs less and appears smaller than at birth, and this even after a large quantity of food, such as it is, has been consumed. The combination of great wasting with a voracious appetite is very striking, and is only apparently contradictory, since hunger—the demand of the tissues for reparative material—cannot be appeased by food which, from its bad quality, is incapable of digestion or proper preparation for absorption and assimilation. Unsuitable food, too, by irritating the mucous membrane of the stomach, creates a fictitious appetite.

Sooner or later the face becomes pinched, the eyes sunken; the lips are pale, and when moved display a deep furrow about the angles of the mouth; the facial expression is uneasy or languid, and the anterior fontanelle is deeply depressed. The skin, generally, is dry, harsh, and yellowish, hangs in loose folds over the bones, and may be mottled by an eruption of strophulus or urticaria, or present red patches of intertrigo in the neighborhood of the genitalia and over the buttocks and inner surface of the thighs. The extremities are cold and the hands claw-like. The tongue is heavily furred or red and dry, and with the mucous membrane of the mouth may be the seat of aphthous ulceration or thrush deposit. As already stated, the appetite is often ravenous, and the cries of hunger are violent, oft repeated, and only temporarily silenced by food; thirst is increased; colic is common; the bowels are constipated, and the stools, which are voided with difficulty and straining, are composed of a few light-colored, cheesy lumps partly covered with greenish mucus.

Attacks of acute vomiting and diarrhœa often interrupt the regular course of the disease. At such times there is moderate fever during the night, though

ordinarily the temperature is subnormal. Again, chronic vomiting and chronic diarrhœa are apt to arise as complications, and greatly increase the danger of a fatal termination.

Sleep is restless and disturbed, and many hours, particularly during the night, are spent in fretful crying. A common group of symptoms connected with the nervous system is "inward spasms." When these occur the upper lip becomes livid, somewhat everted, and tremulous; the eyeballs rotate or there is a slight squint, and the fingers and toes are strongly flexed. They frequently usher in true convulsions.

Sometimes the nervous manifestations are much more complex. Thus, I have seen cases where there was retraction of the head, boring of the head into the pillow, an approximation to the "gun-hammer" decubitus, general hyperæsthesia, and the tache cérébrale,—all suggestive of tubercular meningitis. Such symptoms disappear under an appropriate diet with proper medicinal treatment, and are to be referred to an intensely excitable nervous system—a condition depending upon insufficient nourishment, and differing merely in degree from that leading to "inward spasms."

There is, of course, extreme prostration, the cardiac action is weak, and the respiration shallow. The urine is citron-colored or very dark yellow, has a specific gravity of 1009 to 1012.5, a strong, characteristic odor, and is diminished in quantity. It is always cloudy or milky, only becoming clear on the approach of recovery. The sediment deposited on standing contains variously-shaped cylinders; fatty elements with tinted nuclei; mucus; colored uric acid; urates in a crystallized or amorphous condition; pigment, etc. The reaction is sometimes highly acid. The proportion of urates is decidedly, that of uric acid notably, and of coloring matter and extractives somewhat, increased. Albumin is always present in variable quantity, and sugar also may be frequently detected.

Death may be preceded by convulsions or the symptoms of spurious hydrocephalus, or may result from prostration.

Diagnosis.—Great emaciation may result from inherited syphilis or acute tuberculosis, but both of these conditions are attended by characteristic symptoms, rendering their diagnosis a matter of little difficulty. In inherited syphilis the child snuffles and cries hoarsely. The skin is dry, wrinkled, old-parchment-colored, and mottled with coppery or rust-colored spots. Often the buttocks, perineum, genitalia, and upper portion of the thighs are the color of the lean of ham. Mucous patches are present at the margin of the anus and of the lips. The corners of the mouth are fissured, the nostrils red and excoriated, and the bridge of the nose is flattened. Enlargement of the spleen can frequently be detected on abdominal palpation.

In acute tuberculosis there is fever, the rectal temperature reaching 100° to 101° F. in the evening; cough with irregularly distributed bronchial râles, and usually slight œdema of the legs.

When symptoms resembling those of tubercular meningitis are present, it is often necessary to delay a definite opinion. In simple atrophy, however, the open fontanelle is level or depressed; the belly is never scaphoid; the bowels, though frequently constipated, are never locked; vomiting is apt to be associated with diarrhœa; the respiration and pulse are regular in rhythm; the temperature, as a rule, is subnormal; there is no hydrencephalic cry; and the antecedent history and the course are different from the tubercular disease.

Prognosis.—A vast number of cases die annually in our large cities, yet the results of appropriate management are often rapidly and surprisingly

PLATE XIII.



CASE OF SIMPLE ATROPHY, æt. three months.

Weight at birth, 4 lbs.; weight on admission to Children's Hospital, $3\frac{1}{2}$ lbs. Fed on a mixture of cane-sugar and water.
(Died twelve hours after admission to hospital.)

successful. Patients should never be given up unless there be extreme wasting and prostration, or unless the symptoms of spurious hydrocephalus arise, convulsions occur, or obstinate chronic vomiting or diarrhoea be developed.

Treatment.—For the arrest of wasting from insufficient nourishment, the first and main thing to be attended to is the diet. Without entering at length into this subject,¹ it may be stated, as a uniform rule, that in selecting a diet the object should be to fix upon one suited to the age and digestive powers of the child, so that he may be able to digest, and, therefore, be nourished by, all the food consumed.

Generally, infants under twelve months who have to be either partially or entirely “brought up by hand” do well upon cows’ milk, diluted with lime-water or with barley-water. Often it is well to sterilize the milk, or—a method which has been most uniformly successful in my hands—to add to the milk mixture peptogenic milk powder, and subject to a temperature of 155° F. for six minutes. The food should be administered from a bottle capable of holding half a pint, made of colorless glass, so that the least particle of dirt can be seen, and provided with a soft India-rubber tip. Unless sterilized or Pasteurized, the whole quantity of food intended to be given in a day should never be prepared at once, but each portion must be made separately at the time of administration. Thus, a bottle of the sort described, absolutely clean, may be filled with a mixture of one part of lime-water to two or three of sound milk, or with one part of barley-water to two or three of milk, to either of which may be added from one to two tablespoonfuls of cream and a teaspoonful of pure sugar of milk. The bottle must next be placed in hot water until the contents become warm, when it is ready for the child.

The degree of dilution of the milk and the proportion of cream added vary with the age and feebleness of digestion, but it is upon the latter that we must chiefly base the composition of the food. Lime-water is the preferable diluent when there is frequent vomiting or acid eructation. Both it and barley-water are of service in preventing the formation of large, compact curds—an object that is even better accomplished by peptogenic milk powder, and by the process of partial predigestion. In some cases it may be necessary to discontinue milk foods entirely, putting the child temporarily upon weak broths or raw beef juice.

After digestion has been brought into good condition, the food may be cautiously increased to a standard suitable for a healthy child of the same age. At eight or ten months from two to four fluidounces of thin mutton or chicken broth, free from grease, may be allowed each day in addition to the milk; at twelve months, the yolk of a soft-boiled egg, rice and milk, and carefully mashed potatoes moistened with gravy; and at the end of the second year, a small quantity of finely-minced meat.

Once daily the patient should be bathed in warm water, or at least sponged over with warm water, and every morning and evening a teaspoonful of warm olive oil or of cod-liver oil should be rubbed into the skin over the abdomen and chest. At the same time the belly must be completely covered with a soft flannel binder, and the feet and surface generally kept warm by woollen clothing. In this way attacks of colic, if not entirely prevented, are rendered much less frequent and severe.

If there be intertrigo, cleanliness and the free use of oxide-of-zinc ointment usually suffice to effect a cure.

Of medicines, bicarbonate of sodium, pepsin, pancreatin, nux vomica, and cod-liver oil are perhaps the most useful. Cod-liver oil should not be given

¹ For the details of diet and general management, see Introduction.

until the digestive powers have been brought into a comparatively normal state by proper food, antacids, and digestants and the general tone increased by tincture of *nux vomica*. The oil is most easily borne when given in emulsion, and may be advantageously combined with lactophosphate of lime or with the hypophosphites.

Such symptoms as constipation, diarrhœa, and vomiting demand, of course, appropriate treatment.

DISEASES OF THE CÆCUM AND APPENDIX.

BY JOHN ASHHURST, JR., M. D.,

PHILADELPHIA.

INFLAMMATORY AFFECTIONS OF THE CÆCUM AND APPENDIX.

UNDER the names of typhlitis, perityphlitis, appendicitis, cæcitis, perityphlitic abscess, etc. are included by systematic writers certain cases of inflammation, usually severe and sometimes ending in suppuration or in general peritonitis, met with in the right ilio-lumbar region. While these cases are met with at all ages, they are sufficiently common in children to make their consideration proper in a work devoted to the maladies of childhood, and they are so often attended with danger and lead to such serious consequences that their importance can hardly be overestimated.

The terms *typhlitis* and *cæcitis* are strictly applicable to inflammation, catarrhal or parenchymatous, affecting the cæcum (blind gut) or caput coli; *perityphlitis* to an inflammation of the areolar or connective tissue behind the cæcum, where this portion of bowel is usually uncovered by peritoneum; *perityphlitic abscess* to a collection of pus occurring in the same region; and the somewhat barbarous term *appendicitis* to an inflammation of the pouch or diverticulum known as the appendix vermiformis. Without denying that the caput coli itself may be primarily the seat of inflammation, as indeed may any portion of the intestines, constituting the grave condition *enteritis*, and while acknowledging at least the possibility of a true perityphlitis, perhaps leading to extra-peritoneal suppuration, there can, I think, be no doubt that in the large majority of instances the appendix vermiformis is the part primarily involved, and that the resulting abscess, when pus is formed, is intra-peritoneally situated, though fortunately, in most cases, walled off by adhesions which prevent the general infection of the peritoneal cavity.

Morbid Anatomy.—The pathological lesions found in cases of inflammation of the cæcum and appendix are quite variable. In the majority of cases the inflammation does not advance beyond the stage of lymph-formation, and even after repeated attacks (for the disease is often recurrent) the parts will be found indurated and thickened, and matted together by dense adhesions; but there will be no abscess. In other instances, and particularly when the patient is tuberculous, pus will form at an early period, usually as the result of ulceration and perforation of the intestinal wall, but sometimes without perforation, simply from the intensity of the inflammation. Foreign bodies, such as grape-seeds, etc., are occasionally found lodged in the cæcum or appendix, or loose in the surrounding abscess; but more commonly what are supposed to be foreign bodies are really concretions of earthy phosphates with fecal matter and inspissated mucus, or of inspissated mucus alone. The cæcum from its shape and position is apt to become the lodging-place for concretions of this character, which set up irritation and may lead to ulceration of the cæcal wall, while small

concretions may enter the appendix, or, as is more commonly the case, the mouth of the appendix becoming occluded by catarrhal inflammation and thickening, concretions form *in situ* by inspissation of the retained secretion of the part, which in the normal condition is poured into the cæcum, and forms a natural lubricant for the fæcal mass in its passage through the large intestine. When pus forms in these cases, it may make its way into an adjoining segment of bowel; may become more or less thoroughly encysted and form a fluctuating tumor in the iliac fossa; may burrow in various directions, coming to the surface in the lumbar region above the iliac crest, or, passing downward in the course of the psoas muscle, below Poupart's ligament; or, finally, may infect the general cavity of the peritoneum, causing diffuse purulent peritonitis, which quickly proves fatal. In exceptional cases the pus has been known to perforate the diaphragm, causing pleurisy and empyema, or to enter the hip-joint.

Etiology.—The causes of typhlitis and appendicitis may be divided into the *predisposing* and the *exciting causes*. Among the former may be mentioned *sex*, these affections being much more common in the male than in the female, in the proportion, it is said, of six to one; *age*, most cases occurring in early life; the presence of *tubercle*, tuberculous patients being not only more exposed to appendicitis than the non-tuberculous, but the disease in them more quickly running on to suppuration, and convalescence after an operation, should such be necessary, being effected more slowly and with more interruptions; and *habitual constipation*, the retention of fæcal matter in the cæcum, which is sometimes distended to an enormous size, maintaining a constant source of irritation, and exposing the intestinal wall to the dangers of ulceration and perforation. The *exciting causes* are the entrance of foreign bodies into the appendix—seeds, pins, hairs, etc.; the ingestion of indigestible food; exposure to cold or wet; falls, blows, or strains of the abdominal parietes; and the abuse of drastic purgatives.

Symptoms.—The symptoms of typhlitis and appendicitis are variable and often deceptive. Sometimes beginning with a *chill*, the early symptoms are more often those of enteritis generally, pain, vomiting and constipation, fever, and tenderness with some fulness in the region of the inflamed part. The *pain* is usually greatest in the right iliac fossa, but is sometimes referred to the navel, and may even be most marked on the left side of the abdomen; but even when the pain is misplaced, the greatest *tenderness* will, unless general peritonitis be impending, be found upon the right side, and especially at a point distant an inch or an inch and a half (in the adult two inches) from the anterior superior spinous process of the ilium, and in a line drawn from that point to the umbilicus. This tender spot, which is known as "McBurney's point," corresponds to the position of the appendix, and, as already mentioned, it is the appendix which is primarily involved in the large majority of cases. At a later period, when pus has formed, the "soft spot" which precedes pointing of the abscess may sometimes be detected in precisely the same locality. Coincidentally with the development of tenderness in the right iliac region, gentle palpation will reveal a *fulness*, followed at a later stage by tenderness and tumefaction, in the position of the cæcum; and in order to relieve the inflamed part from pressure of the superjacent tissues, the patient will usually secure relaxation of the abdominal wall by lying on his back, slightly turning to the right side, and with the right knee drawn up.

The *vomiting* is often distressing, attended with considerable effort, and aggravates the pain by succussion of the inflamed parts: the ejected matters consist at first of the contents of the stomach, and afterward of the intestinal juices with bile; fæcal vomiting does not, as a rule, occur, even when general

peritonitis follows, this being a point of some importance in the diagnosis of these conditions from intussusception and other forms of mechanical obstruction of the bowel. The *constipation* in appendicitis and typhlitis is not complete: there may be an occasional discharge of flatus; evacuations may be secured by the use of enemata, and the administration of salines may cause even free catharsis without modifying the other symptoms of the disease. The *fever* is not very intense, the temperature varying from 101° to 102° F., and is accompanied with a quick pulse, furred tongue, and intense thirst: when suppuration occurs the fever may assume a hectic type, and in the cases which terminate unfavorably the tongue becomes brown and dry, sordes accumulate about the lips and teeth, and the patient passes into a decidedly "typhoid" condition.

When *suppuration* occurs the symptoms undergo some modification. The pain and tenderness are usually increased; rigors may occur at irregular intervals; the tumefaction in the right iliac region becomes somewhat boggy, the overlying integument being perhaps congested and slightly œdematous; a "soft spot" may be observed; and, if the pus be not evacuated, fluctuation, with ultimately pointing, as in abscesses elsewhere. There are sometimes pain in the right knee and ankle, and œdema of the leg. The pus in these cases commonly has a strong faecal odor from proximity to the bowel, even though no perforation be discoverable.

Diagnosis.—The diagnosis of appendicitis and typhlitis can usually be made without difficulty if the symptoms be carefully noted, the affections in regard to which confusion is most likely to occur being enteritis, intestinal obstruction, psoas and iliac abscess, and hip disease. *Enteritis*—by which term is meant inflammation involving all the coats of a segment of intestine—is well described by Sir Thomas Watson as "peritonitis with something more." It may occur in any part of the bowel, not being limited to the right iliac region, and the localizing symptoms of appendicitis—McBurney's point, etc.—are therefore wanting. The paralysis of the gut is more complete, constipation consequently being more absolute, with no discharge of flatus, and the vomiting, if relief be not afforded, soon assuming a faecal character. Typhlitis, using the term accurately, is of course a form of enteritis, but when the inflammation is limited to the cæcum the symptoms are less severe than when a larger portion of bowel is implicated. *Mechanical obstruction* of the intestine in children is usually of the character of *intussusception*, though *internal strangulation* by bands or diverticula is occasionally met with. In the latter condition the pain would be felt mainly at the seat of obstruction or more commonly at the umbilicus; there would be no fever, the temperature more probably being subnormal, sometimes even after the development of peritonitis; there would be faecal vomiting, with absolute constipation and inability to pass wind; general tympany, from paralysis of the bowel allowing gaseous distention; partial suppression of urine; and the patient would pass into a state of collapse, sooner or later according to the position and closeness of the strangulation. In *intussusception* there might be fever from secondary inflammation of the affected bowel; there would be a tumor, but instead of occupying the right iliac fossa, it would be found in a median position or upon the left side; there would, in acute cases, be a discharge of blood and mucus from the bowel; and digital exploration of the rectum would, in children at least, probably detect the lower end of the invagination. *Psoas abscess* is usually, though not invariably, accompanied by evidences of preceding disease of the spinal column, is not attended by pain or marked tenderness, and presents no intestinal complications; *iliac abscess*, if depending upon ovarian or periuterine inflammation and

situated on the right side, may more closely simulate appendicitis; but even here the distinction may be made by observing the absence of bowel symptoms. In *hip disease* the peculiar and characteristic deformity and malposition of the limb, varying with the stage of the disease, will suffice, when present, to clear up the diagnosis; in appendicitis, though extension of the limb may cause great pain, it is not accompanied by the arching of the lumbar spine observed in hip disease, and the joint may be moved, without causing suffering, in other directions. In the rare cases in which an abscess, originating in appendicitis, opens into the hip-joint, causing secondary disease of that articulation, the symptoms would be confused, both maladies then, in fact, coexisting in the same subject; but under ordinary circumstances the absence of intestinal symptoms in the one case, and the absence of joint symptoms in the other, ought to prevent the possibility of error.

With regard to the special diagnostic importance of "McBurney's point," a good deal of difference of opinion prevails among practitioners, and the tendency at the present time is to consider it of but little value. For my own part, I am disposed to place considerable reliance upon this symptom, and believe that the detection of induration and tenderness, or at a later period of a "soft spot," in this particular situation is, while perhaps not pathognomonic, at least strongly significant of disease originating in the appendix.

Tumor of the kidney, perinephric abscess, carcinoma of the bowel, and abscess of the abdominal wall have been mistaken for appendicitis, but careful examination and investigation of the history of the case ought to prevent an error in this direction.

The diagnosis of perforation of the cæcum or appendix may be made when symptoms of suppuration occur, or when the spread of pain and tenderness to the left side of the abdomen indicates the threatened implication of the peritoneum generally. Fortunately, before or immediately after the occurrence of perforation, adhesions usually form and seal off the affected part from the rest of the peritoneal cavity, and even where this does not occur, an interval of some hours, or even a day or two, may intervene before the development of universal peritonitis, giving an opportunity for prompt surgical intervention which may save life even in this emergency.

Prognosis.—The prognosis in appendicitis and typhlitis is in the large majority of cases favorable. Under judicious treatment the acute symptoms will subside in from four days to a fortnight, although a certain amount of induration and tenderness may persist for a much longer period. The patient is now apt to become intolerant of the regimen and rest which has been hitherto enforced, and resumes his ordinary diet and manner of living, with the result that relapse occurs; and this sequence of events may be repeated indefinitely. The reason that recurrence of appendicitis is so often met with is, I believe, that the patients will not persist in treatment until completely recovered. If thoroughly cured, a second attack is not, according to my experience, to be particularly dreaded.

When perforation occurs the prognosis becomes more gloomy. In the rare cases, if such exist, in which the opening is in the cæcum behind the peritoneum, a burrowing abscess will result, and convalescence will, under the most favorable circumstances, be tedious. If the perforation be intra-peritoneal, peritonitis, local or general, is inevitable; in the former case, the infected area being separated by adhesions from the general cavity, recovery after operation may be hoped for; in the latter, though by prompt intervention a patient may occasionally be snatched, as it were, from the very jaws of death, yet the large majority will perish; diffuse suppurative peritonitis is almost always a fatal

affection. In tuberculous patients the prognosis, *cæteris paribus*, is always less favorable than in others.

Treatment.—The treatment of appendicitis and typhlitis may be either prophylactic or curative. As *preventive* measures, care should be taken to avoid constipation by regulation of the diet, by encouraging defecation at a fixed hour daily, and, if necessary, by the use of laxatives. The patient should be warmly clad, especially around the abdomen, should keep the feet dry, and should avoid exposure to cold and wet generally. When the disease actually occurs, the indications for *remedial* treatment are—(1) to keep the inflamed part at rest; (2) to relieve the congestion; (3) to prevent pain; and (4) to maintain the patient's nutrition without overtaxing the impaired powers of digestion. If suppuration occur, the pus must be promptly evacuated by incision and drainage. The first indication is met by keeping the patient in bed and by avoiding the use of purgatives, which under these circumstances can only do harm. The constipation and consequent accumulation of faecal matter in these cases are owing to paralysis of the bowel, more or less complete, due to its inflamed state; or, in other words, are a *result*, not a *cause*, of the inflammation. This is a distinction which often the friends of the patient, and sometimes even the physician, seem unable to comprehend; they cannot understand that the patient is not ill because his bowels are not moved, but that his bowels are not moved because he is ill. In saying this I am not unmindful of the fact that salines, in small but frequently repeated doses, are often used in these cases, and that the patients sometimes do well under this treatment; but the benefit is due to the action of the remedy as an indirect means of effecting depletion and drainage, and if this could be accomplished without catharsis it would be so much the better. The second and third indications are met by the application of leeches (if the symptoms are very urgent), and by the use of warm cataplasms and the administration of opium. The fourth indication is met by careful feeding with peptonized milk or other liquid nutriment, or, if the patient vomit, by employing nutritive enemata. The course of treatment may then be established as follows: The patient being strictly confined to bed, a few leeches are applied over the seat of greatest pain, drawing from two to six fluidounces of blood according to his age; if for any reason leeching be thought inadvisable, a small blister may be applied, and the part afterward covered with mercurial and belladonna ointments, equal parts, spread upon lint, and over this in turn a warm flaxseed or elm poultice. Enough opium should be given to relieve pain, either by the mouth in the form of the deodorized tincture, or by suppository; or morphia may be given hypodermatically if preferred. Belladonna may properly be combined with the opium, and is also to be used locally with the mercurial ointment, as already described. When the pain has entirely ceased, but not before, if the bowels do not move spontaneously in the course of twenty-four hours, a warm enema of olive oil and soap-suds may be administered; if this fail, and if there be no tendency to vomiting, small doses of the Epsom or Rochelle salt—from half a drachm to a drachm—may be tentatively given every hour or two hours, the enema being repeated twice daily; if there is nausea or vomiting, the saline should be omitted, and calomel in minute doses (gr. $\frac{1}{20}$ — $\frac{1}{12}$), with bicarbonate of sodium (gr. j-ij), may be given instead. Administered in this way, and the patient being still kept under the influence of opium, I doubt if these medicines cause any increase of peristalsis, and the good which they undoubtedly do is, as already mentioned, due to the serous flow from the congested and inflamed bowel to which they give rise.

After the subsidence of all acute symptoms the salines may be continued in

reduced doses, so as to cause two or three passages from the intestines daily, and the local use of mercury and belladonna, or a belladonna plaster, should be continued until the swelling and tenderness have disappeared, when the remaining induration may be treated by painting the part with tincture of iodine every day or every other day, according to the effect produced, maintaining mild but persistent counter-irritation without blistering. During the early stages the right lower limb may be flexed over a pillow to relax the abdominal wall, but as soon as possible it should be brought flat, and, if there is any tendency to permanent contraction, weight-extension should be applied to keep the limb in proper position.

In the large majority of cases, unless the patient be tuberculous, prompt and persistent treatment on the lines above indicated will suffice to effect recovery. After convalescence the patient should live by rule, avoiding indigestible food, and observing all the precautions referred to in speaking of prophylaxis.

If, however, instead of yielding to treatment, the symptoms persist, and the evidences of deep-seated suppuration—fluctuation, superficial œdema, or a “soft spot”—are manifested, no time should be lost in resorting to an exploratory operation. So important is promptness under these circumstances that it has been maintained that in every case the physician should associate with himself a surgeon to watch the patient from the beginning of the attack, so that there may be no delay when the critical moment arrives. I am not prepared to say that this is always necessary, but I do say that if a physician undertakes the management of a case of appendicitis alone, he should possess the *tactus eruditus* which will enable him to recognize suppuration as soon as it occurs. I have more than once been called to patients who had been treated many days, if not weeks, by practitioners who had not detected the presence of pus, the signs of which were yet, to the surgical sense, quite obvious.

OPERATIONS FOR APPENDICITIS.—It was formerly recommended, when suppuration was believed to have occurred in cases of cæcal or appendiceal inflammation, to verify the diagnosis by the introduction of an exploring needle; but the feeling of modern surgeons is against the use of this instrument, as being very apt, on the one hand, to miss striking the purulent collection, and, on the other hand, if it should reach the abscess, apt to infect the peritoneal cavity as it is withdrawn; and a careful incision of moderate extent is, I have no doubt, safer in every way than the blind thrust of a needle-point, as well as more likely to discover the seat of suppuration. Before making the incision the abdominal wall should be thoroughly cleansed and purified, but with great care and gentleness, as it would be quite possible for a vigorous antiseptic scrubbing to break through the limiting adhesions and diffuse the contents of an abscess through the peritoneal cavity. Operators differ as to the best line for incision: when it was believed that the purulent collection was formed outside of the peritoneum, the rule, as laid down by Willard Parker, Hancock, Buck, and Sands—who may be regarded as the pioneers in this branch of surgery—was to make the incision above Poupart’s ligament, as in tying the external iliac artery, and endeavor to reach the abscess by cautiously working upward and pushing the serous membrane out of the way; but since it is now generally recognized that, as taught by Weir, the abscess is actually intra-peritoneal in origin, the marginal incision is no longer thought important, and surgeons aim to reach and evacuate the pus by the most direct route. If the case is so far advanced that fluctuation is manifest, the incision should be made where this is most perceptible; but under other circumstances the best position, I think, is in the general direc-

tion of the right linea semilunaris, taking care that a part of the wound shall be through the so-called "McBurney's point," which, as already mentioned, corresponds to the usual situation of the appendix. Some operators prefer to place the incision more laterally, believing that they thus secure better drainage, but, upon the whole, in most cases, I prefer the anterior position.

The first cut, about four inches in length, should pass through the skin and superficial fascia, and the deeper layers are then cautiously divided upon a director, all bleeding being checked before the abdominal cavity is opened. When the peritoneum is reached, it is cautiously raised with forceps and nicked by the edge, not the point, of the knife held sideways—as in the operation for strangulated hernia—the wound being then carefully enlarged with blunt-pointed scissors guided and guarded by the finger as a director. As soon as the cavity is opened a gush of pus will usually serve to confirm the diagnosis, but if this does not occur the surgeon should cautiously explore with his finger and a blunt director in the neighborhood of the cæcum until the seat of suppuration is discovered. After evacuation of the pus the cavity is carefully but thoroughly washed out with hot distilled water, and the surgeon then searches for the appendix, which, if found, should be removed. Often this can be done without difficulty, the organ, enlarged and thickened, being readily separated by the finger from its adhesions and brought out at the wound; its neck should then be tied with two strong carbolized silk ligatures, and divided between them. If, however, the appendix cannot readily be found, it is better to allow it to remain than unduly to prolong the operation by hunting for it, nothing being more deleterious in abdominal surgery than prolonged delay and unnecessary manipulation of the viscera.

After a final washing with hot distilled water, a full-sized drainage-tube, of glass or rubber, should be introduced, carried to the bottom of the cavity, and secured with a stout ligature or safety-pin. Some surgeons merely pack the wound with iodoform gauze, instead of introducing a tube, but my own preference is for the latter practice. As to the choice between glass and rubber, my rule is, when the abscess-cavity is completely walled off from the general peritoneal surface, to use a rubber tube, which is shortened from time to time as the wound heals; but when the peritoneal cavity is opened, I employ a glass tube, armed with a rubber-dam and containing a rope of absorbent cotton, which is renewed as often as it is saturated without disturbing the dressing applied to the rest of the wound, the tube being at the same time sucked out with a long-beaked syringe until the secretion becomes of a pale straw color, and is reduced to a minimum, when the tube is finally removed. A few sutures may be applied to the extremities of the wound, but it should not be tightly closed, being rather allowed to heal firmly by granulation and cicatrization.

There is little or no danger of consecutive hernia in this situation, and if there is any communication with the bowel, fæcal fistula will be less apt to follow in an open wound than in one which has united only superficially. Fæcal fistula, however, contrary to the doctrine formerly held, is really a rare complication after the operation for appendicitis, and is not to be dreaded unless some grave constitutional condition, such as general tuberculosis, interfere with the healing of the wound.

All surgeons are agreed as to the propriety of operative intervention in cases of acute appendicitis in which suppuration is believed to have occurred, but some go further, and enthusiastic operators advise that the appendix should be removed after recovery as a means of preventing recurrence of the disease. I have myself operated under these circumstances, and successfully, but I think

that there are very few cases in which such a course is justifiable. The time to perform an operation in itself dangerous—and opening the peritoneal cavity is dangerous, gynæcological and surgical enthusiasts to the contrary notwithstanding—is when a greater and imminent danger may be averted by so doing, and not when the patient is well; and when we consider that the very extensive statistics of the London Hospital show that 90 per cent. of all cases of appendicitis end in recovery without operation, we may well hesitate before submitting a patient to a mode of treatment equally needless and heroic. The only circumstances which seem to me to justify an operation after convalescence are when the patient has had repeated attacks at decreasing intervals and of increasing severity, and when he is going to be so placed that skilled surgical assistance will not be available in the event of further recurrence.

NON-INFLAMMATORY AFFECTIONS.

The cæcum has occasionally been found in a hernial protrusion (*cæcal hernia*), as has the appendix, the latter particularly in the variety of rupture incorrectly called congenital, in which the bowel escapes into the patulous vaginal process of peritoneum. Cæcal hernia is often irreducible through the formation of adhesions between the portion of gut uncovered by peritoneum and the adjoining structures. The appendix, even when not itself diseased, sometimes acquires adhesions to other parts, and may then cause *internal strangulation*, a loop of bowel being caught beneath the appendix and constricted as if by a fibrous band. Should such a condition be discovered during an operation for intestinal obstruction, the appendix should be divided between two ligatures, or, which would be better, excised, so as to prevent the possibility of a recurrence. *Malignant growths* are met with in the cæcum, though not often in children, and may be treated on the same principles which guide the surgeon in dealing with similar affections in other portions of the bowel.

INTUSSUSCEPTION.

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INTUSSUSCEPTION, or invagination of the bowel, is by far the most frequent cause of mechanical obstruction of the intestine met with in childhood, though internal strangulation by an adherent appendix or by Meckel's diverticulum, or more rarely by a band of organized lymph left from a previous peritonitis, occasionally occurs. Invagination, as the name implies, consists in an ensheathing of one segment of bowel within another, the invaginated part being almost always from a higher portion (that is, farther from the anus) than that into which it is received. Thus, the jejunum is invaginated into the ileum, that into the cæcum and colon, etc. The much rarer condition, that in which the lower segment is received into the upper, is called *retrograde intussusception*. It is not uncommon for this affection to occur among the multiple invaginations which arise during the act of dying, but *direct intussusceptions* are those which are met with during life, and which call for treatment. Every complete intussusception involves three layers of bowel, and each layer consists of all the intestinal coats; the outer layer is the *sheath*, or *receiving layer*, the *intussusciens*; and the internal or *entering layer*, together with the middle or *returning layer*, constitutes the invaginated part, or *intussusceptum*. The *apex* of the intussusception is at the junction of the inner and middle layer—the lowest point, therefore, of the intussusceptum; while its neck is at the junction of the middle and external layers—the uppermost part of the intussusciens. *Double intussusceptions* are occasionally met with, five layers of gut being then involved, either a second intussusceptum having been forced into the first, which then constitutes its sheath, or the intussusciens with its contained intussusceptum being in turn invaginated into a fresh portion of bowel, which then forms a second sheath. Still more rarely *triple intussusceptions*, involving seven layers of bowel, have been found.

Locality.—In rather more than one-half of all cases of intussusception the invagination occurs about the junction of the small and large intestines: usually the cæcum, and afterward the colon, is inverted, the ileum pushing before it the ileo-cæcal valve, which is thus found at the apex of the intussusceptum; much more rarely the ileum slips through the valve, which then constitutes the neck of the intussusciens, and the intussusceptum grows by successive invagination of fresh portions of small intestine. The former variety is known as *ileo-cæcal*, and the latter as *ileo-colic intussusception*. In somewhat less than one-third of the whole number of cases the invagination is limited to the small intestine (*ileal* or *jejunal intussusception*), and in the remainder, or about one-sixth, to the large intestine (*colic intussusception*).

Except in the ileo-colic variety, in which the neck remains fixed, an intussusception increases at the expense of its sheath, which becomes gradually inverted, the apex of the intussusception remaining constant while its neck is

continually changing; in the ileo-colic variety fresh portions of ileum keep passing through the valve, and the neck therefore remains unchanged while the apex varies. As the entering layer carries the mesentery with it into the sheath, a certain amount of traction is exerted upon one side of the intussusceptum, and as a result the intussusception becomes curved or even sharply flexed upon itself, and at the same time the apex becomes displaced toward the mesenteric side of the intussusciens, both of these conditions tending mechanically to render the occlusion of the gut more complete than it would be otherwise. The *extent of bowel* involved in an intussusception varies from a few inches to six or more feet.

Though an invagination usually begins on the right side of the abdomen, its increase, in the most common or ileo-cæcal variety, is mainly at the expense of the large intestine; and therefore by the time it has acquired sufficient size to be recognized by palpation the *tumor* will be chiefly on the *left* side, and eventually the ileo-cæcal valve with the apex of the intussusceptum may be protruded from the anus: even when this does not occur, the apex, in children at least, can very commonly be detected by digital exploration of the rectum.

Morbid Anatomy.—The adjacent serous layers in an intussusception soon become more or less closely united by adhesions, which, if firm, render the invagination *irreducible*. These adhesions may join the two layers of the intussusceptum to each other over a considerable space, or may be limited to the region of the neck; they are very seldom found exclusively at the apex. The sheath of the intussusception may become ulcerated from pressure, and even perforation may occur; but more commonly, beyond a certain amount of congestion and inflammation, no marked changes are found in this layer. The intussusceptum, on the other hand, is usually more or less completely strangulated, and becomes gangrenous, when, if there are firm adhesions at the neck, the dead portion may be separated and evacuated as a whole or in segments through the anus, the patient eventually recovering. Under other circumstances, the adhesions being defective, fæcal extravasation into the peritoneal cavity may occur, the death of the patient following; or the adhesions, while preventing death at the moment, may form the starting-point of a stricture, which in turn, at a later period, may cause fatal obstruction.

Post-mortem inspection in a case of intussusception reveals the elongated tumor caused by the invagination, usually on the left side of the abdomen, with an apparent absence of that portion of bowel which is invaginated. The outer layer or sheath of the intussusception is usually of a gray color, doughy in feel, and sometimes ulcerated from distention, while the intussusceptum, when exposed, is found of a deep-red color, resembling a clot of blood, or black and gangrenous. The intestine above the seat of obstruction is commonly much dilated, and filled with fæcal matter and gas, while that below is collapsed and shrunken, and is either empty or contains a small quantity of blood and mucus. There is sometimes general peritonitis.

Etiology.—Nothnagel has investigated experimentally the causes of intussusception, and describes a *paralytic* and a *spasmodic* variety, the latter being the more frequent. Differing from the ordinary doctrine, he believes that the invagination is caused by the normal gut being drawn over the spasmodically contracted part, rather than by that being mechanically driven into its sheath. Treves also adverts to the influence exercised by the longitudinal muscular fibres of the bowel, acting from the contracted part as from a fixed point, and thus drawing the uncontracted part over the other. Age and sex are usually spoken of as *predisposing causes* of invagination, the large majority of cases

occurring in male children; the great relative length of the colon in infancy, together with the width of the mesocolon, doubtless favors the displacement of the gut, and in some degree accounts for the frequency of intussusception in the early periods of life. Impaired general health, diarrhoea, the presence in the bowel of undigested or irritating food, polypoid growths, strictures and tumors of the intestine, and previously existing adhesions, are often predisposing causes of more or less importance. The *exciting cause* is increased and irregular peristaltic movement, no matter how produced.

Symptoms.—The chief symptoms of intussusception are pain, nausea and vomiting, tympanitic distention of the abdomen, fever, tenesmus, with discharge of blood and mucus by the rectum, the presence of a tumor (usually on the left side), and a corresponding depression or flattening on the right side. Abdominal *pain* is usually the first symptom manifested, occurring suddenly, of a very intense character, referred mainly to the umbilicus, the child writhing and drawing up its limbs in agony, and accompanied by vomiting of whatever may be in the stomach, and often by a liquid faecal discharge, evacuating the contents of the bowel below the seat of obstruction. The pain is not constant at first, but occurs at irregular intervals, each paroxysm being commonly attended by a discharge of bloody mucus from the rectum, but as the case goes on the pain becomes continuous, though even then marked by exacerbations. The cause of the pain is at first the mechanical squeezing of the invaginated bowel by its sheath; afterward the increased peristalsis of the intestine above, endeavoring to force its contents through the part which is occluded; and finally, the extreme distention of the upper bowel and the inflamed condition of the intussusception itself and of its peritoneal covering. A sudden cessation of pain in the last stages indicates the occurrence of gangrene, which may be followed by discharge of the sphacelated portion and recovery, but is more often the immediate precursor of death. Abdominal *tenderness*, localized at the seat of invagination, is developed in connection with the pain as soon as inflammation of the affected portion of bowel has set in.

The *vomiting* in intussusception is a very prominent symptom, being present, according to Dr. Fitz's statistics, in 70 per cent. of all cases, but is, I think, less distressing, in the early stages at least, than in cases of internal strangulation. When secondary enteritis occurs the vomiting increases, but even then comparatively seldom assumes a faecal character. The vomiting diminishes again with the approach of collapse.

Tympanites is not very marked in intussusception, being, according to Dr. Fitz, only present in the minority of cases. Indeed, there is often a marked depression in the right iliac fossa (*signe de Dance*) from the displacement of the caecum toward the left side.

Fever is not present at the beginning of an intussusception, but is observed in connection with the occurrence of secondary enteritis, the thermometer rising to 102° or 103° F. This is of some importance in aiding the diagnosis between invagination and internal strangulation, the temperature in the latter condition sometimes remaining subnormal even after the development of general peritonitis. *Partial suppression of urine* often accompanies the fever in intussusception, and appears to depend more on the acuteness than on the locality of the disease.

Unlike other forms of intestinal obstruction, invagination is not necessarily accompanied by *constipation*, though in the acute variety, owing to the lateral displacement of the gut from traction of the mesentery and to secondary enteritis, faecal discharges are absent. In chronic intussusception, however, there may be little interference with defecation, and in acute cases there is a

constant desire to go to stool (*tenesmus*), with frequent discharges of blood and mucus. This symptom Mr. Pollock considered to be almost pathognomonic.

The tumor is a very characteristic symptom of intussusception, and, as already mentioned, is usually found on the left side. In this it differs from the tumor of fecal impaction, which is almost always found on the right side, and which may often be made to pit by deep pressure over its surface. The right side in intussusception is, as mentioned above, often depressed and flattened (Dance's sign), and the tumor is painful and tender to the touch. It can frequently be detected by introducing the finger into the rectum, and sometimes comes so low as to protrude from the anus.

Chronic intussusceptions are sometimes met with, and have been particularly studied by Rafinesque, who finds that 70 per cent. occur in the region of the ileo-cæcal valve (60 per cent. ileo-cæcal, 10 per cent. ileo-colic), and that the remainder are equally divided between the large and small intestine. The symptoms of chronic invagination are much less distinctive than those of the acute variety, the tumor changing its shape and locality from time to time, fecal evacuations being often continued, diarrhœa sometimes alternating with constipation, and the pain and vomiting occurring at perhaps long intervals.

Diagnosis.—Intussusception has been confounded with simple colic, appendicitis, enteritis, dysentery, fecal impaction, and other forms of mechanical obstruction. From *colic* it may be distinguished by the paroxysmal character of the pain, the vomiting, and the tenesmus, with discharge of bloody mucus. The detection of a tumor, either on the left side of the abdomen or by rectal exploration, would further demonstrate the nature of the affection. From *appendicitis* and consequent *suppurative peritonitis*, the diagnosis can be made by noting the symptoms just referred to, and by further observing that in those affections there are tympanites, tenderness, and fulness in the right iliac fossa (as contrasted with the depression in invagination), and an earlier development of fever. In *enteritis* there is also fever from the beginning, with constipation, but without bloody discharges and without any well-defined tumor. I have known the convexity of the lumbar vertebræ, as felt by abdominal palpation, to be mistaken for the tumor of intussusception, but the error could hardly be made except by carelessness. The tenesmus, pain, and evacuation of blood and mucus are the only points of resemblance between intussusception and *dysentery*, while the mode of attack and course of the several affections are entirely different. In *fecal impaction* there is a tumor, but usually on the right side, and it can be indented by firm pressure, while the peculiar evacuations of invagination are wanting. The only form of *mechanical obstruction*, apart from intussusception, which is likely to be met with in children is *internal strangulation*, and in that condition the profound and early collapse, the low temperature, and the stercoraceous vomiting will clear up the diagnosis.

Prognosis.—The prognosis in cases of intussusception is always grave in the extreme, Leichtenstern's statistics showing a death-rate (in acute cases) of 73 per cent., and Fitz's smaller figures one of 69 per cent. The most favorable termination is in spontaneous reduction of the invagination, which can, as a rule, only be effected during the first few days of the attack, before the formation of adhesions. If reduction fails, there remains a chance for recovery after sloughing of the intussusception, the mortality in cases in which this occurs being only 41 per cent. while in those in which sloughing is absent the death-rate is 85 per cent. Even when sloughing does occur, however, and the patient recovers from the immediate risks of the process, he is by no means free from the danger of ulterior complications, the cicatricial contraction and adhesions

which follow often, as already mentioned, laying the foundation for future obstruction by stricture or internal strangulation.

The prognosis of *chronic intussusception* is also very grave: while the immediate risks to life are less than in the acute cases, there is not the same hope of recovery by sloughing and evacuation of the invaginated part, and, unless relief be afforded by an operation, a fatal result must be anticipated.

Treatment.—The indications for treatment in *acute intussusception* are to put the bowel completely at rest; if the case is seen at an early period, to attempt reduction; and, if the invagination has already become irreducible, to sustain the patient's strength until separation of the strangulated part may occur, when recovery may be hoped for. The first indication is met by the free use of opium, preferably in combination with belladonna. These remedies are best given in the form of the extract, by suppository, and of the former one-twelfth of a grain, and of the latter one-twenty-fourth, may be administered to a child of two years, every hour or two hours according to the urgency of the symptoms. Morphine and atropine may be used hypodermatically instead, but the rectal administration is, on the whole, I think, to be preferred. Advantage may also be derived from the employment of anodynes locally, and the abdomen may be covered with belladonna and mercurial ointments spread upon lint or flannel and reinforced by a warm poultice. In the attempt to effect reduction the physician may employ large *injections* of warm water, or, which is, I think, better, warm olive oil; *inflation* with atmospheric air or various gases; and manipulation or *abdominal taxis*.

The *injections* may be given with an ordinary hand-ball syringe or with a fountain syringe (gravity injection), the patient being etherized and held in a semi-inverted position, with the hips higher than the shoulders, and the trunk elevated at an angle of about 45°. The height to which the reservoir which supplies the fluid should be raised will be about eight feet in the case of an infant, and not more than twenty feet in that of an adult. The quantity to be injected may vary from one to six quarts according to the age of the patient. The injections are best administered through a large rectal tube, so that the force of the current may, if possible, be directed immediately upon the apex of the intussusception, and not expended upon the wall of the bowel. Care must be taken not to allow the fluid to escape alongside of the tube, by providing this, as suggested by Mr. Lund, with an India-rubber collar, which may be firmly pressed against the anus, or by wrapping it with cotton or lint, which is introduced within the sphincter to accomplish the same end.

Inflation with atmospheric air may be practised through the long tube or long-nozzled bellows, the same precautions being taken against escape of the air alongside of the tube as in the use of enemata. Professor Senn recommends the employment of hydrogen gas as preferable to atmospheric air, the gas being supplied from an India-rubber balloon holding four gallons, which is slowly but steadily compressed by the operator. Carbonic-acid gas is preferred by Libur, Jate, and Ziemssen, and is furnished in a nascent state by successively injecting solutions of bicarbonate of sodium and tartaric acid. *Abdominal taxis* was introduced as a mode of treatment in these cases by Mr. Jonathan Hutchinson, and consists in systematically compressing and kneading the belly from below upward, the patient being etherized and in an inverted position. In combination with the use of enemata it has occasionally proved an efficient remedy, but its employment is necessarily attended with some danger of injury to the bowel, and should therefore, it seems to me, be resorted to with caution, and only during the early stages of the case.

Reduction by one or other of the methods mentioned is most likely to be

accomplished during the first two days of an intussusception, and may occasionally be effected as late as the fourth day, but after that period should not be attempted, the physician's efforts being then directed to sustaining the patient through the processes of sloughing and evacuation of the strangulated intussusception. In this stage the use of opium and belladonna should be continued; little or no food should be given by the mouth, but the patient should be systematically fed by means of nutritive enemata. To relieve thirst, which is often distressing, water may also be given by enema, and the patient may suck small pieces of ice. If the abdomen becomes very much distended, the stomach may be carefully *washed out* through a stomach-tube, thus allaying vomiting and evacuating the liquid contents of the upper portion of the small intestine; or gas and fluid may be withdrawn by puncturing a distended segment of bowel with the fine tube of an aspirator. *Puncture of the bowel*, practised in this way, entails a certain risk of fæcal extravasation, but is followed by less shock than enterotomy, which, however, may be preferred when the patient's condition does not forbid it.

Enterotomy—or, as it is sometimes called, Nélaton's operation—consists in making an incision, usually in the right iliac region, and opening the first distended coil of intestine which presents itself. This may be done in two ways: if it is not desired to establish a false anus, a knuckle of bowel is gently drawn out through the wound, and, having been packed around with sterilized gauze, is opened, preferably by a transverse incision, and allowed to discharge itself outside of the abdominal cavity; if the evacuation is not sufficiently complete, a full-sized drainage-tube may be introduced into the gut, and the surgeon sits by the patient, keeping the bowel under observation, if necessary, for several hours, until the fæcal flow has entirely relieved the tension; the tube is then removed, the opening in the intestine closed with a Lembert's suture, the bowel replaced, and the external wound closed and dressed in the ordinary manner. If it be thought better to establish temporarily a false anus, the bowel should first be stitched to the abdominal parietes, then carefully opened, and the edges of the incision again stitched to the external wound so as to prevent any possibility of fæces escaping into the cavity of the peritoneum. If the case does well, after the separation and evacuation of the gangrenous intussusceptum the false anus may be allowed to close, as it usually will without difficulty as soon as the natural passage is restored. If the opening degenerate into a fæcal fistula, a plastic operation may be required for its relief.

The mode of treatment above described is that which I would recommend in cases of *acute* intussusception. *Laparotomy*, which may be required in cases of *chronic* invagination, does not seem to me desirable in cases of the acute variety, and is shown by statistical investigation to have no effect in diminishing the death-rate of the disease. Thus, while Leichtenstern's collection of 557 terminated cases, taken all together, gives 151 recoveries and 406 deaths (73 per cent.), the tables published in the fifth edition of my *Surgery* give 95 cases treated by laparotomy, with 26 recoveries and 68 deaths (1 undetermined), showing an almost identical percentage of mortality. Fitz's statistics present the operation in a still less favorable light, 51 cases treated without operation having given 16 recoveries and 35 deaths (69 per cent.), while 36 operated on gave only 6 recoveries and 30 deaths (83 per cent.). The objections to the operation in acute cases are that there is, as has been seen, a reasonable chance of recovery without it, and that the early age at which intussusception usually occurs renders operative interference peculiarly dangerous. I am well aware that a few brilliant results from laparotomy in infants

have been recorded by Mr. Hutchinson, the late Dr. Sands, of New York, and other operators, but these cases should be regarded as surgical curiosities, showing what infants may sometimes safely endure, rather than as furnishing precedents for future guidance. In *chronic intussusception* the circumstances are somewhat different. As the strangulation of the intussusceptum is not sufficiently complete to offer a chance for recovery by the process of sloughing, when the surgeon finds that reduction cannot be effected the operation may be properly resorted to, particularly as in these cases the patients have usually passed the period of infancy. When the bowel protrudes through the anus, the plan suggested by Howse, and successfully employed by Mikulicz, Willard, Fuller, and others, may be tried, the protruding portion being held from retracting by strong pins, and then cut off; but under other circumstances laparotomy is the proper measure.

Laparotomy for intussusception may be thus performed: The patient having been etherized and the abdominal wall carefully cleansed, an incision is made directly over the tumor if one can be recognized, but otherwise in the median line. The wound is carefully deepened until the peritoneum is reached, when this is opened with every precaution against injury to the bowels or other viscera. If the intussusception is found, the invaginated gut is brought out through the incision, the rest of the intestine being gently pressed back with warm towels or sponges, since the exposure and chilling of large portions of bowel always produces an unfavorable effect on the patient. Careful attempts at reduction are then to be made by gently compressing and pushing upward the invaginated part from below, this being at once safer and more efficient than efforts to withdraw the gut by traction from above. If the intussusception is not immediately found, the surgeon introduces his hand, through the incision, which in this case would be median, and explores the right iliac fossa, as recommended by Mr. Treves, finding the cæcum, and then searching upward or downward according as that part is empty or distended with fæces. In examining the small intestine the direction in which the search should proceed may be determined, as suggested by Mr. Head, by observing the relations of the mesentery, which is attached to the posterior wall of the abdomen from the left side of the second lumbar vertebra, obliquely downward to the right sacro-iliac symphysis. If reduction cannot be effected, the surgeon may proceed to the establishment of a false anus immediately above the seat of invagination, or, if the state of the patient should permit more prolonged manipulation, he may excise the intussusception bodily (*enterectomy*), and restore the continuity of the bowel either by direct suture (*circular enterorrhaphy*) or by Prof. Senn's method of *lateral anastomosis*, as may be thought best. The latter procedure or one of its modifications—for a description of which the reader is referred to special works on surgery—is ordinarily preferable, as requiring less time than the end-to-end suture. The subsequent treatment is to be conducted as after laparotomy for other causes, as has been described in the article on Diseases of the Appendix.

INTESTINAL PARASITES.

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THE older writers on the diseases of children devote a good deal of space to the subject of intestinal worms, particularly to the symptoms supposed to be caused by them, and to their treatment. Text-books of to-day dwell more upon the natural history of these animals—an extremely interesting subject—but are apt to pass very lightly over the practical considerations of symptomatology and treatment. Although intestinal worms, like the teeth, have with propriety been dethroned from their high position as etiological factors in many of the diseases of children, we must not be carried too far with the swing of the pendulum and disregard entirely the parasite as a causative agent. Among the laity, with exceptions among the upper classes, worms still hold a very important position, and it is essential, therefore, that we should look at the subject fairly, and not pass it off as of very minor importance.

There are no intestinal parasites peculiar to infancy and childhood, although the round- and pin-worms are so much more common in children than in adults that they are often spoken of as peculiar to children.

Omitting several varieties that are rarely encountered and are of no practical importance, the species of worms that are found in children are as follows: *Ascaris lumbricoides*, round-worm; *oxyuris vermicularis*, pin-worm; two species of tape-worms, *tænia mediocanellata*, beef tape-worm, and *tænia solium*, pork tape-worm; and the unimportant *trichocephalus dispar*. All of these are Nematode worms, with the exception of the *tæniæ*, which belong to the group of Cestodes.

As these parasites have different habits and habitats, and each requires a special treatment, it will be necessary to consider them individually.

I. *ASCARIS LUMBRICOIDES* (ROUND-WORM).

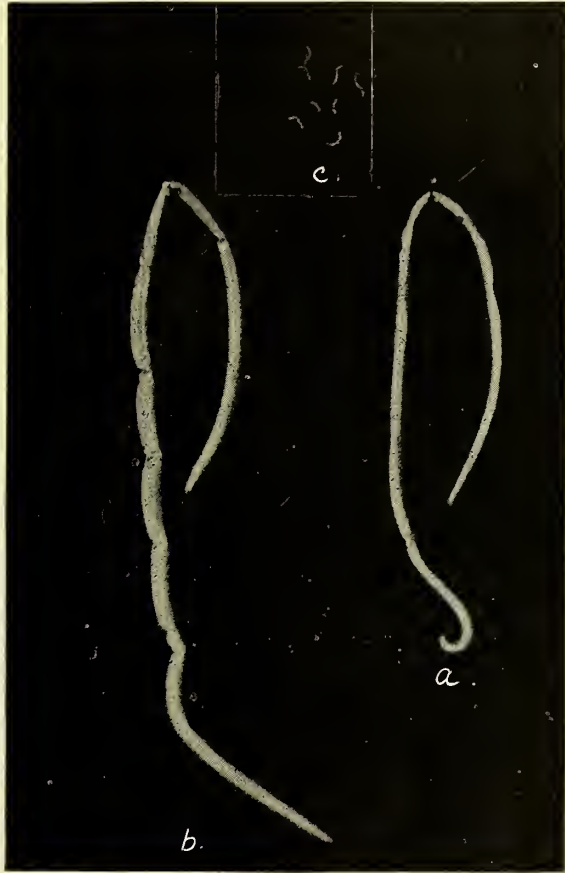
The male round-worm is from four to six inches in length, the female about ten inches. It is of a yellowish-white color, more or less tinged with red in the fresh state; as usually shown, preserved in alcohol, it is of an ivory white. The worm is cylindrical in shape, tapering to a point at both ends. The mouth is situated between three lips furnished with fine teeth at the anterior extremity of the body. The anus is about an inch from the posterior extremity, and the vulva in the female is anterior to the middle. The sexes are easily distinguished by their relative size and by the fact that the posterior extremity of the male is curved, that of the female being straight (Fig. 1, *a* and *b*).

From earth-worms, which I have known to be presented by patients with the intention of deceiving, they may be distinguished by their color and by the fact that earth-worms, being annelids, have plainly-marked segments. Female lumbricoids which have been carelessly handled and subjected to pres-

sure often show the ovaries hanging out like a bunch of small worms, and may deceive the superficial observer.

The ova of the round-worm are produced by the females in great quantities,

FIG. 1.



Round-worms and Pin-worms ($\frac{2}{3}$ Natural Size). *a*, Male Round-worm: *b*, Female Round-worm; *c*, Female Pin-worm.

and pass off in the fæces, where they can easily be found with the microscope. They are oval in shape, about $\frac{1}{400}$ of an inch long, with dark granular contents and thick transparent coats, which are often stained yellow by bile (Fig. 2, *c*).

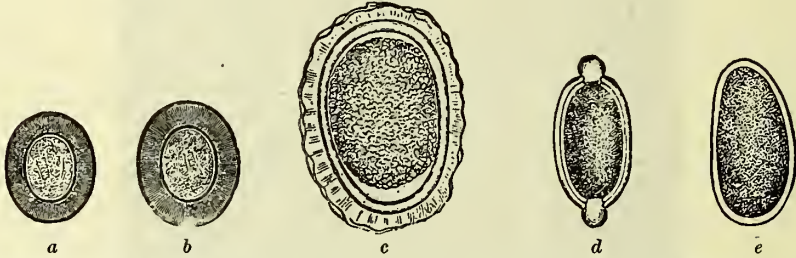
The proper habitat of the adult ascarides is the small intestine, but they are of a wandering disposition, and have been found in the stomach, œsophagus, and mouth, occasionally getting up into the posterior nares and coming out anteriorly, or going down into the larynx or even into the lungs. They also wander down into the rectum, and are expelled with the fæces or slip out unattended. They have even escaped into the peritoneal cavity through perforations made, not by them, as was once supposed, but by ulcerations. They have been known to pass into the pancreatic and biliary ducts. When in large numbers the worms are often coiled together into balls in the intestines. The ova do not develop until they have passed out with the fæces, and have again found their way into the child's gastro-intestinal canal, when the embryos

rapidly come to maturity. Outside of the body they resist destructive agencies with great obstinacy, and it is said may retain their vitality for years.

Method of Infection.—As the ova are produced in such countless numbers—Davaine having found some three thousand eggs in a bit of feces as large as a grain of wheat—and as they are so resistant to outside destructive agencies, it is not surprising that they should be very common among the classes of individuals where personal cleanliness is not cultivated. As children are greater barbarians in their personal habits than adults, it is natural that ascariides should be much more often found among them. The habit children have of putting their fingers as well as toys and other objects into their mouths might easily lead to self-infection with ova from parasites in their own intestines, as well as with ova from elsewhere. In the country, infants creeping about the floor may be infected by the dust brought in on the shoes from manure-heaps.

Among the upper classes ascariides are certainly very much less common, and they are rarely seen in the adult. Here, where habits of cleanliness are

FIG. 2.



Comparative size of eggs of Intestinal Parasites: *a*, *Tænia Solium*; *b*, *Tænia Mediocanellata*; *c*, *Ascaris Lumbricoides*; *d*, *Trichocephalus Dispar*; *e*, *Oxyuris Vermicularis*. (After Strümpell.)

cultivated, infection would be more likely to come only through drinking-water or food. If the contents of privies are used in the garden for manure, the contained ova may readily find their way into water used for drinking or be served with salads or other uncooked vegetables. By proper filtration of the water or by cooking of vegetables, this danger can be escaped.

Symptoms.—It is not uncommon to find numerous intestinal worms in the lower animals without any evidence of ill effect, and it is frequently the case that we discover lumbricoides, in greater or less numbers, in the dejections of children who are well in every way and have presented none of the classical symptoms of worms. It is certainly the case, therefore, that, while the round-worm is confined to its proper place—the small intestine—even if it be in great numbers, it may be, and generally is, entirely harmless, and has no appreciable effect on the condition of the child, producing no symptoms. The amount of nourishment it extracts for itself is hardly worth considering unless the worms exist in great numbers.

On the other hand, when we consider the high state of nervous tension that exists in the child, and the ease with which reflex phenomena are produced, it is reasonable to suppose that the presence of the living worms in the intestine may cause certain reflex symptoms, and in that way interfere with the general health.

The common symptoms ascribed to round-worms by the laity are general lassitude, with nervous fidgeting, picking at the nose, offensive breath, abdominal pain, headaches, feverish attacks—called “worm fever”—and lack of flesh, notwithstanding a fair, or at times ravenous, appetite. The bowels are irregular, there being either constipation or diarrhœa with mucous discharges. There

may be vomiting and disturbed sleep with grinding of the teeth. This is the common and exact picture of a child debilitated by improper feeding and an insufficiency of fresh air and exercise—a child that is cooped up with many others in close school-room air, and whose whole life is poorly managed from a hygienic point of view. That such children sometimes have ascarides is not surprising when we consider the ease of infection, but that the parasites are the cause of their condition is certainly not the case, although the nervous symptoms may undoubtedly be aggravated by them. It is probable that these debilitated children, with plenty of mucus in their intestines, are more desirable habitats for the round-worms, so that the parasites thrive in this class and retain their foothold, while healthier children more easily get rid of them.

The symptom, picking the nose, is often spoken of by mothers as if it were pathognomonic of worms. This is not the case; it is simply a nervous trick common to debilitated children, as is often proved by the unproductive administration of anthelmintics. My experience is that in the majority of cases where round-worms are found, their presence is unsuspected and their discovery accidental. Having once been found, it is common enough for almost any symptom to be attributed to them by the mother.

Numerous cases have been reported, however, where the connection between the worms and severe nervous symptoms, such as convulsions, chorea, aphonia, etc., seemed to be very intimate, the nervous symptom being relieved on the evacuation of the parasites.

One such case is recorded among those in the Boston Children's Hospital:

Kate M—, four years old, had had two convulsions before she came under observation. She was in good general condition, and no reflex cause could be found for the convulsions except round-worms, which she had passed from time to time. She was given *santonin*: a quantity of worms were expelled, and she remained well for six months, when she had another convulsion. Worms were again brought away, but she came back a month later, reporting occasional attacks of twitching and tremors, but no real convulsions. Anthelmintic treatment again expelled round-worms, and she was lost sight of for three years, during which there was no history of worms or nervous phenomena. At the end of this time she again applied for treatment for attacks every two or three weeks of flushing and pallor, pain in the belly and convulsions. *Santonin* was again given, bringing away worms and giving relief as before.

There is a certain mechanical danger from ascarides, owing to their habit of wandering. A number of cases have been recorded of these worms entering the cystic and common bile-ducts, giving rise, in the latter case, to jaundice. They have even penetrated to the hepatic ducts and caused abscesses of the liver. They have also been found in perityphlitic, hernial, and tubercular abscesses connected with the intestine, having wandered into these abscesses after their formation, and possibly in some cases contributing to the irritation and suppuration there. That they may cause perforation of the normal intestine is not the case, but when we consider their stiffness and activity during life, and their sharply-pointed extremities, it does not seem unlikely that they might break through an ulceration which needed only the last straw, so to speak, but which otherwise might have healed.

Another danger from round-worms arises from the fact that they sometimes ascend—with or without the aid of vomiting—into the fauces, whence they may be drawn into the larynx and cause suffocation and death. If the worm be drawn into the trachea or a bronchus and is not expelled, death is not immediate, but ensues in three or four days from gangrene of the lung. The fact that a child is found dead with a lumbricoid in the larynx does not, however, necessarily prove that this was the cause of death, for these worms

not infrequently wander away from the intestine after death from other causes. In the majority of cases when the worm ascends to the fauces it is expelled through the mouth, or more rarely, through the nose.

When the parasites are collected in great numbers in the intestine, they may mechanically cause congestion of the mucous membrane, and even obstruction of the bowel, or volvulus. In these cases the worms are found tightly twisted together, forming an obstructive ball.

Hillyer, in the *Lancet* (1892, ii., p. 773), relates an interesting case of this sort, where there were at the same time extreme nervous symptoms:

A child, five and a half years old, never strong, began to have severe abdominal pains, for which castor oil was given with the result of causing vomiting, but no action of the bowels. Three round worms were found in the vomitus. The child then became unconscious, the eyes wide open, the pupils dilated, the skin cold and clammy. Death ensued on the following day. At the autopsy the ileum was found occluded at a point fifteen inches above the ileo-caecal valve by a tightly-wound ball of eight round worms. Forty-two worms in all were found in the intestine. There was extreme congestion of the intestine above the obstruction and at that point. Below the obstruction the bowel was empty, above it was distended.

Diagnosis.—This can never, and should never, be made without seeing the worms themselves or their eggs. Mothers in their anxiety often mistake shreds of mucus for worms, so it is essential that the physician should see the suspected parasites in every case. As was stated above, debilitated children with mucus in their dejections are the ones that present symptoms popularly thought to be diagnostic of worms.

The ova are so numerous that they are easily found in the sediment of liquid stools; this can be scraped from the napkin or taken up with a pipette, or the residue examined after filtration. If the stools are not naturally liquid, they can be stirred up with water. A method suggested by Epstein is simple and effective,—viz. the introduction of a Nélaton catheter into the rectum. The small amount of fæces that will cling to the eye of the catheter is more than sufficient for microscopic examination. The power generally used for urinary sediments—*i. e.* about 330 diameters—answers for these examinations. The eggs, which have been described above, are easily recognized (Fig. 2, *c*), and readily distinguished from the smaller, sharper, oval eggs of the pin-worm and the round eggs of the tape-worm.

Treatment.—Although ascarides, as a rule, cause no discomfort and are in no wise detrimental to the host, when we consider the various accidents, some of them fatal, which may be caused by them, as well as the obscure nervous symptoms which occasionally owe their origin to this source, it is certainly wiser to treat all cases as soon as they are discovered, and to get rid of the worms.

Of the remedies that can be used for round-worms, it is hardly worth while to mention more than three. These are *santonin*, *spigelia*, and *chenopodium*. All of these have the power of killing or benumbing the parasites, but require the aid of cathartics to cause their expulsion.

Santonin, made from Levant worm-seed, is probably the most widely used of all anthelmintics. It is the common basis of proprietary worm-lozenges. Care should be used in its administration, as it is extremely poisonous in overdoses, several fatal cases having been reported. In poisonous doses it produces gastro-intestinal irritation, dizziness, tremor, yellow vision, dilated pupils, and loss of consciousness, with, at times, convulsions. *Santonin* is an almost tasteless white powder, nearly insoluble in water. It can be given in powder mixed with sugar, or made up into lozenges. The dose at the age of two years is

$\frac{1}{4}$ to $\frac{1}{2}$ grain; at six, 1 grain; and at twelve or fifteen, 2 grains. It should be given morning and night, or in some cases three times daily, with the addition of a cathartic—calomel, castor oil, or cascara cordial—every second day as long as lumbricoids continue to be passed. When it is remembered that very grave symptoms have been caused by a dose of 4 grains to a child four years old, and that a feeble child of five has been killed by 2 grains of santonin, it is easily seen that care must be used in its administration, and that there is danger in its indiscriminate use.

Spigelia, or pink-root, one of our native plants, is also an efficient and, in proper doses, entirely safe drug. The freshly prepared fluid extract of spigelia and senna¹ of the Pharmacopœia of 1870 combines the necessary cathartic with the anthelmintic in a manner both efficient and pleasant to the taste. The dose is half a teaspoonful for a child of two years, a teaspoonful for one from four to ten years old. It should be given two or three times daily, depending on its effect upon the bowels.

Oil of chenopodium is the third remedy for ascariides, and is said to be safer and less irritating than the others. It can be given on sugar in doses of five drops to a child of three, and ten drops to one of ten years, three times daily. A cathartic is required, as with the other anthelmintics, and should be given every second or third day.

II. OXYURIS VERMICULARIS (PIN-WORM, THREAD-WORM, SEAT-WORM).

This is a small worm, as the first two of its common names would imply. (Fig. 1 shows the comparative size of pin- and round-worms.) The female is from a quarter to half an inch in length; the male, only about a third as large, measures from $\frac{1}{12}$ to $\frac{1}{8}$ of an inch. Its color is nearly white, its shape fusiform, tapering to a fine point in the female, having a blunter and generally curved tail in the male. The mouth is situated in the middle of the blunt end, and is surrounded by three slightly projecting lips (Figs. 3, 4). The eggs are ovoid in shape, more pointed at one end. They measure 0.053 mm. in length by 0.028 mm. in breadth, are considerably smaller than the eggs of ascaris, and have a thinner and smoother coating (Fig. 2, e).

This worm inhabits the rectum and large intestine throughout its entire course, as well as the lower end of the small intestine. The eggs are passed out with the feces in great numbers, and, when swallowed, the embryo is set free in the digestive tract and descends to the colon, rapidly developing into the adult worm. The number of these parasites in one individual may be so enormous that the whole mucous surface of the colon and rectum becomes coated with them, as if with a layer of pus. In the cæcum, where they are undisturbed, the sexes are about equally divided. In the rectum and in the stools the females preponderate, as, owing to their larger size, they are less easily destroyed than the smaller more fragile males. The great preponderance of females is also partly apparent, as the males, from their minute size, are often overlooked. Pin-worms are frequently seen alive outside the anus in the folds of skin, sometimes getting into the groins, and in little girls they often crawl into the vagina.

Method of Infection.—Auto-infection is constantly taking place in children having pin-worms. The irritation caused by the worm leads them to scratch about the anus; numerous eggs become lodged under the finger-nails, and are later taken into the mouth and stomach. It is very common to find

¹ This can be written for directly. Its formula is as follows: *R.* Ext. spigeliæ fl., f 3x.; Ext. sennæ fl., f 3vj.; Olei anisi, mxx.; Olei cari, mxx.

several children in one family suffering simultaneously. Food and toys that are handled by these children become carriers of the infection. Vegetables and drinking-water may also be infected, as in the case of round-worms.

Symptoms.—The oxyuris gives rise to a very evident symptom in nearly all cases—namely, an intense itching about the anus, which leads the patient



FIG. 3.
Oxyuris Vermicularis: a, Male, Natural size; b, The same enlarged. (after Beneden).

to scratch vigorously, causing bleeding and in some cases setting up an eczema. The itching occurs most severely in the early part of the night while the patient is in bed. It is thought to be due to the movements of the worms in the rectum, and is entirely relieved by their removal from this point, even if they remain in quantities higher up. In fact, it is probable that the parasites while in the small intestine produce no appreciable symptoms.

As a result of the itching the sleep of the child is disturbed, and various slight nervous symptoms may be induced. Grinding of the teeth, crying out in sleep, involuntary twitching, and insomnia are common. In one of my cases pavor nocturnus was apparently caused by the reflex irritation of the worms; and in a very sensitive child it is probable that reflex convulsions or chorea might ensue. As a result of the disturbed rest and of the more or less constant irritation, the patient is often debilitated, peevish, and nervous, and, like all nervous children, apt to acquire the trick of picking the nose, and to have occasional reflex feverish attacks. He may, however, escape without a symptom.

In two of the pin-worm cases at the Boston Children's Hospital fainting was a prominent symptom. One of these, a girl of ten years, was said to have "worm-fever" about once a month. At this time she had fainting attacks and passed great quantities of the parasites.

As a reflex cause of incontinence of urine these parasites hold a well-recognized place. In eight of the hospital cases incontinence existed. In girls vulvo-vaginitis is sometimes caused by the irritation of the worms that have found their way into the vagina; this, in turn, is also a cause of incontinence of urine. Nine instances are recorded among 48 cases at the hospital. As 34 of these cases were in girls, this makes a proportion of 27 per cent. of vulvo-vaginitis from this cause. The great preponderance of females in this list, 34 to 14, may be partly explained by the urgent symptom of vulvo-vaginitis calling them to the hospital for treatment. Curiously enough, the same preponderance of girls is also found in the round-worm cases—11 girls to 5 boys. Masturbation in either sex may be caused by the irritation. One of my cases had a rectal polypus, probably due to rectal irritation. Prolapse of the rectum may be set up by the straining. As to the age at which these worms are chiefly found in children, 35 of the 48 cases at the hospital occurred in children between two and seven years old, inclusive. The youngest was an infant of twenty-one months.



FIG. 4.
Oxyuris Vermicularis: a, Female, Natural size; b, The same enlarged. (after Beneden).

As bearing on the frequency of worms in general, and of each species in particular, I have examined the out-patient records of the Boston Children's Hospital, and find that out of 5200 medical patients of all kinds, there were 65 where the diagnosis of worms was made on the evidence of the parasites themselves. My general impression was that the round-worms were more common than the pin-worms in children, and this is so stated by Councilman in the *Cyclopædia of the Diseases of Children*. A much larger number of the latter were seen at the Children's Hospital, however, owing no doubt to the more urgent symptoms they produce, and the general absence of symptoms in round-worms. Forty-eight of the 65 cases had pin-worms, and only 17 round-worms, 3 of these being afflicted with both varieties. The remaining four had tape-worms—in 1, *Tænia solium*; in 1, *Tænia mediocanellata*; and in 2 the species was not accurately determined.

Diagnosis.—As in all cases of intestinal parasites, the diagnosis can only be made with certainty by the discovery of the worm itself or the ova. The history of anal pruritus in a child should always lead one to suspect the presence of pin-worms, and the anus and its neighborhood should be carefully searched. By the use of an enema large numbers of the worms may be brought to light. By examining under the microscope scrapings from beneath the finger-nails, the folds about the anus, or the detritus scooped out from inside the anus with a grooved director or catheter, the eggs are often found in large quantities, and are easily recognized, as described above. In all cases of incontinence of urine, masturbation, and leucorrhœa the oxyuris should be thought of and sought for.

As in the case of lumbricoids, intestinal mucus, which in greater or less quantity is mingled with fecal discharges, has often been mistaken by the nurse or mother for pin-worms, as is illustrated by the following case:

Allen M—, three and one-half years old, was brought to my clinic at the Boston Children's Hospital with the history of having passed great quantities of pin-worms in the last few days. His symptoms, which the mother attributed to the worms, were vomiting, slight diarrhœa, with feverishness and general debility. He had a similar attack a year ago, and was thought to have passed worms then. Examination in the folds about the anus failed to reveal any worms, and a microscopical examination of detritus from under the finger-nails, outside the anus and inside the anus was negative as regards the finding of ova. The mother brought next time some of the feces which she believed to be swarming with the worms. The fecal mass when placed in water showed plenty of stringy mucus, which, gathered in thread-like clusters, certainly simulated very closely actual pin-worms. There was in this case undoubted irritation of the intestine, giving rise to various symptoms suggestive of worms, and to an extra secretion of mucus. The irritation, however, was due to an improper diet, not to worms.

Treatment.—As long as any worms remain in the bowel there is a constant source of infection. Treatment must therefore result in the complete expulsion of the parasites, or we shall have, what is often unfortunately the case, a relapse or return of the trouble. Besides this, measures must be taken to prevent reinfection from the old sources after cure. If the worm confined itself to the rectum, as is erroneously believed by some, treatment from below with injections would be simple and effectual. This treatment, although giving relief for a time, is of course entirely inadequate, as many of the worms are out of reach in the cæcum or even in the lower part of the small intestine. The proper method, therefore, is to make the attack both from above and below. By the mouth may be given either *santonin*, *spigelia*, or *chenopodium*, with a cathartic, in the manner already described in the treatment of *ascarides*. Cathartics which produce free watery discharges are found to be particularly efficient in the treatment, even without a previously administered vermicide.

Epsom salts, Seidlitz powder, or Hunyadi water are therefore to be recommended, but are all unfortunately distasteful to children. The syrup of raspberry disguises very well the taste of Epsom salts in a 25 per cent. solution, thus:

R_x. Magnesii sulphatis ʒiv.
Syrupi rubi idæi fʒij.—M.

Sig. A tablespoonful containing one drachm of the salts.

The vermicide and cathartic may be given by the mouth two or three days in the week.

Once a day the rectum should be washed out with a copious enema of cool soapy water. By using a soft-rubber catheter attached to the nozzle of the syringe the enema can be introduced higher up, and will be more effectual. Plenty of water should be used, so as to distend the folds of the rectum and colon in which the worms are lodged. Cold water alone is effectual in washing out and killing the worms, but the addition of castile soap makes it less irritating to the bowel and more fatal to the worm; and this addition is all that is necessary if the injections be given thoroughly. Other substances are often used in solution in the enema for their destructive effects on the worm. These are common salt, quinine, quassia, alum, tannin, etc., but it may be doubted whether these solutions are any more effectual than properly given injections of soap and water. Where there is relaxation and protrusion of the rectum an astringent injection is of use, as, for example, one drachm of sulphate of iron to one pint of infusion of quassia; or a solution of tannin can be given, in the proportion of a heaping teaspoonful to a pint of water. All irritating injections should be avoided, and dangerous ones, like solutions of corrosive sublimate, had better not be used.

As the worm or its ova may live in the folds about the anus, these parts should be carefully scrubbed with soap and water and anointed with an antiseptic ointment. Boric-acid ointment, as in the following prescription, besides destroying worms, is of use in allaying the irritation or eczema caused by their presence:

R_x. Acidi borici ʒj.
Olei rosæ gtt. iij.
Vaseline ʒj.—M.

Even after a complete cure, obtained by the expulsion of all the worms, reinfection is likely to take place unless certain precautions are taken. The bed-clothing, the blankets, as well as the linen, may contain the eggs of the oxyuris; the toys undoubtedly have some lodged in their crevices; and the carpet or floor may be more or less infected, for it must be remembered that a small bit of fecal matter spilt from a vessel or napkin may contain thousands of eggs. The room and its contents should therefore be almost as thoroughly cleaned as in the case of one of the exanthemata. The bed-clothing should be boiled, the toys destroyed, the carpet and rugs thoroughly beaten, and the floor and furniture scrubbed with soap and water. The neglect of this undoubtedly accounts for the frequent failures to cure this troublesome affection.

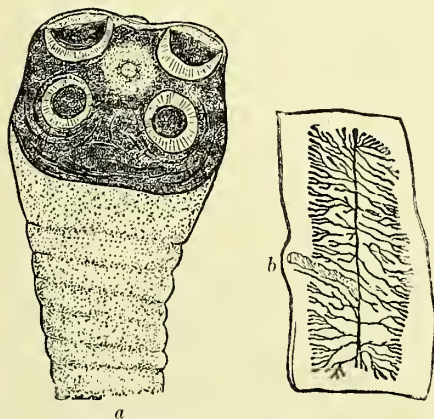
III. TÆNIA (TAPE-WORM).

THE common tape-worm is from twenty to fifty feet in length, of a white color, and composed of numerous flattened segments, each of which, except

those near the so-called head, is a complete hermaphrodite. Nourishment is absorbed through the body-walls from the contents of the intestinal canal, in which the whole worm lies immersed. The "head" is a modified segment about the size of the head of a pin, and it is by this organ with its suckers or hooks that the worm retains its hold on the intestine. The segments near the head are not much broader than a piece of thread, but they rapidly increase in size and become from one-quarter to one-half an inch broad at the other extremity of the worm.

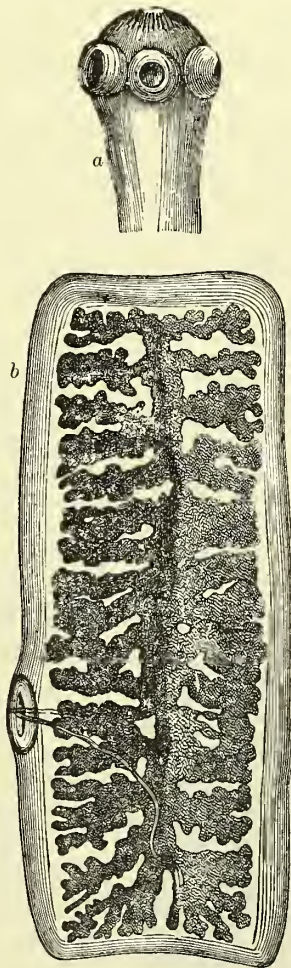
Varieties.—The two species commonly found in this country are the beef tape-worm, *Tænia mediocanellata*, and the pork tape-worm, *Tænia solium*.

FIG. 5.



Tænia Mediocanellata. Head and Mature Segment, Enlarged (Heller).

FIG. 6.



Tænia Solium. Head and Mature Segment, Enlarged. (Beneden.)

Two other species may be mentioned, as they are sometimes encountered: *Tænia nana* and *Tænia cucumerina*. Another species, belonging to a different genus, *Bothriocephalus latus*, is found only in certain parts of the continent of Europe.

The beef and pork tape-worms (Figs. 5 and 6) are easily distinguished by their heads, and less readily by the sexually mature segments. The pork tape-worm has a circle of hard chitinous hooks on the head, with four sucking disks, and the head itself is somewhat pointed. The head of the beef tape-worm is not pointed, and is provided with four suckers only, being devoid of the circle of hooks. This species may also be distinguished by the sexually mature segments or proglottides which are passed from the anus. In the pork tape-worm the lateral branches of the uterus (Fig. 6, *b*), are only eight to twelve in number, and quite thick, while in the beef tape-worm the side branches are finer and are much more numerous, being twenty or thirty in number (Fig. 5, *b*). These can be seen by flattening out the segments between two microscopic slides and holding them up to the light. The addition of glycerin makes them more transparent.

Tænia nana has of late years been found to be very numerous in Italy, particularly in Sicily. It has been found in Egypt, and also in England. With so many Italians of the poorer class constantly coming to this country, its occurrence here is to be expected. It especially attacks children, and may occur in great numbers in one individual. It is very small, being only ten to fifteen mm. in length. The head is armed with four suckers and a rostellum with hooks, which can be protruded or entirely withdrawn. Severe nervous symptoms are sometimes caused by this worm.

Tænia cucumerina is another rare form of tape-worm which especially infects children, being acquired by them from dogs.

Life History.—The ova are produced in each segment in great numbers, and those of the two common varieties of *tænia* are easily distinguished from the eggs of the round and thread-worms by their smaller size and spherical instead of oval shape. The eggs of *T. mediocanellata* are slightly larger than those of *T. solium*, which are about $\frac{1}{750}$ of an inch in diameter (Fig. 2, *a* and *b*).

The tape-worm lives in the small intestine, firmly attached to the mucous membrane by the suckers and hooks on its head. While the head is attached to the upper part of the jejunum, the other extremity, in the common species, may reach nearly or quite to the ileo-caecal valve. The pork tape-worm is generally found singly, while two or more beef tape-worms may occur in the same individual. The worm grows by a process of breeding or segmentation from the segments close to the head. As these become farther and farther removed from the head by this process, they become larger and sexually mature. The first sexually mature segment of *T. solium* is about the four hundred and fiftieth from the head. Some of the ova are extruded from the lower mature segments, and pass off with the feces, but most of them escape from the anus still contained in the ripe segments, which break off entire. These segments, besides passing out in the fecal mass, may slip out of the anus into the under-clothing; and this happens so frequently that attention is usually called to the presence of the worm in this way.

For the development of the eggs another host is utilized, this host being the hog in the case of *T. solium*, and cattle in the case of *T. mediocanellata*. In the case of the hog, with its fondness for grubbing around in heaps of offal and manure, infection easily takes place. Cattle may be infected in a similar way while cropping grass that has been fertilized with human feces. In the animal's stomach the thick outer coatings of the ova are dissolved, the embryos are set free, and proceed at once to pierce the stomach-walls, and, carried along in the blood-current, bury themselves in the muscles, the liver or other viscera. Here they develop into cysticercus cysts, which in the pork tape-worm are a little larger than a pea, in the beef tape-worm somewhat smaller. Within these cysts the larval *tænia* or scolex grows, the head being formed with a short neck and a flask-shaped body (Fig. 7). These cysts remain quiescent for from three to six years, after which they die and become calcified. If, however, the flesh containing living cysts is taken into the human stomach, the larval scolex sprouts into the mature tape-worm and the cycle of changes is complete.

It occasionally happens that the eggs of tape-worms are swallowed by men, and cysticerci may develop in various parts of the body, especially in the subcutaneous and intermuscular connective tissue, or in the brain or eye.

Method of Infection.—The consumption of raw or imperfectly cooked



Cysticercus, or larval tape-worm.

meat, in which the temperature has not been raised to a sufficient point to kill the cysticeræ, is the source of infection for tænia. Infants and children are liable to become infected with the beef tape-worm from the use of raw meat, sometimes recommended in intestinal troubles. When the beef is very finely minced or when the juice only is used, the beef being thoroughly pressed and strained, this danger is removed. The consumption of raw sausages is a more common cause of the pork tape-worm among continental nations than in this country; here the beef tape-worm is probably more commonly met with.

Children, from their uncleanly habits and their custom of sucking the fingers, are more exposed to the danger of swallowing the ova and developing cysticeræ.

Symptoms.—The symptoms caused by tape-worms in the intestine are as obscure as those of round-worms, and, as with these parasites, are often lacking. A child, as well as an adult, may harbor a tape-worm for years, the only indication of this being the passage of segments from time to time *per anum*. Uncomfortable sensations in the abdomen and pain in the region of the navel, with the various nervous symptoms given under the head of Lumbricoids, such as picking at the nose, disturbed sleep, fitful and at times ravenous appetite, have been observed in these cases. There may be nausea and salivation, and vomiting is at times present. The bowels are often irregular. The movements of the worm in the intestine are sometimes described, but it is doubtful whether this is anything more than a psychological phenomenon. Failure to take on flesh notwithstanding a ravenous appetite is to the laity a characteristic symptom of tape-worm, but its significance is of very doubtful value, for it is a symptom often present without the worm, and is indicative of faulty digestion and imperfect assimilation. As with the other intestinal worms, chorea and convulsions have been attributed to tæniæ.

The following case came under my care at the Children's Hospital in July, 1890:

Angelina M——, four and a half years old, has had a tape-worm for two years. The mother has found segments frequently in her under-clothing and in the stools. She has been under treatment by various doctors from time to time, but without permanent relief, as the whole worm has never been expelled. The child was accustomed to eat a great deal of very rare beef. She complains of constantly feeling tired, is peevish and fretful, frequently picking the nose, and is restless at night. Her head perspires a great deal, her appetite is at times ravenous, and she complains of pain about the navel. The bowels are regular.

Under treatment—which I shall give below—she expelled a beef tape-worm twenty-four feet long, with the head entire. It is extremely interesting to note that a year later, in May, 1891, the patient returned, complaining of exactly the same symptoms, which had never been recovered from, but she never passed any more segments of worm.

Diagnosis.—There is no difficulty in making the diagnosis of tape-worm, for the mature segments slip from the anus at intervals of every few days or are passed in the stools. Their white color and peculiar shape at once attract attention, so that it is not necessary to make microscopic examinations of the feces or to resort first to anthelmintic treatment. The distinction between the two common species of tænia is made in the manner detailed in the description of the worms. The fact that patients are apt to mistake shreds of mucus for worms, requires the physician to assure himself of the correctness of the identification before beginning treatment.

Treatment.—Having made sure that a worm is present, appropriate treatment should be at once instituted unless contraindicated by some acute illness;

for, although the worm in the intestine may produce no symptoms, there is always danger of cysticerci developing somewhere in the body from the accidental ingestion of the ova.

Half-hearted measures are sure to be failures, consuming time, irritating the child, and wasting its strength. To be successful the entire worm, including the head, should be obtained, although it often happens that if the worm be broken off close to the head and expelled, there is no return of the trouble. This can probably be explained by the fact that the head is in reality expelled, but, being so small, is not found in the faecal débris. This is particularly apt to be the case if the mother or nurse attempt to find the head. It is much better for the physician himself to make the search. This should be done by adding water to the stool and shaking up the faecal mass or stirring it gently with a stick, being careful not to break up the worm; by decanting the water from time to time and adding fresh, a clear mixture will be obtained in which it is easier to find the parasite.

Treatment consists, first, in the preparatory dietetic management; secondly, in the administration of some drug which experience has shown will kill or benumb the worm; and lastly, in the use of a cathartic to remove the offending body.

The preparatory treatment is partial starvation, in order to weaken the worm. For this purpose small amounts of such food as can be digested in the stomach are to be preferred, and the colon should be unloaded, so as to make the exit for the worm easy. As children cannot stand much starving, the preparatory period should be shorter than in adults, and it loses some of its irksomeness by including the night. After a light dinner the child should be given a bowl of beef-tea with a half slice of white bread for supper; an enema must be given in the evening and the child put to bed early. The breakfast must consist of beef-tea alone. An hour later, say at 9 A. M., the anthelmintic can be given, to be followed in one hour by the cathartic. The stools should be carefully preserved and examined as explained above. It sometimes happens that the worm is partially expelled by a movement from the bowels, and is left hanging out of the anus. In this case great care should be used not to break it off, a large injection being given to dilate the rectum and allow the removal of the worm by gentle traction. Dilatation of the anus by a small rectal or nasal speculum will take off the pressure of the sphincter and aid in extraction.

It only remains to consider the various tænicides recommended. The list is a large one, but I will mention only the important ones. These are—pomegranate, the bark of the root and its alkaloid pelletierine; filix mas, the root of the male fern; kousso; pumpkin-seed; turpentine; and cocoa-nut.

The first, pomegranate, is one of the most efficient. It can be given in a decoction, which, however, makes a disagreeable draught, and one apt to defeat its own purpose by causing vomiting. A much neater way, and one that I have always employed, is to use the alkaloid pelletierine. The tannate of this alkaloid is made into an elegant but very expensive preparation by Tanret of Paris, and is put up in small bottles containing one adult dose. This can be obtained in all our large cities, and its efficiency makes up for its high price. As pomegranate in full doses causes nausea, giddiness, faintness, and indistinctness of vision, it is best for the child to lie down after the dose is given. In the case of tape-worm in the child of four and a half years, related above, the preparation of the tannate of pelletierine was given, one-third of the bottle, which contained five teaspoonfuls, being administered at a dose. The child complained of slight dizziness and headache. An hour after the tænicide a full

dose of castor oil was given, and four hours later the worm was expelled entire.

The oil of male fern, *oleoresina aspidii*, is the next most efficient remedy for tape-worm, a teaspoonful being given to a child of five years, shaken up with some agreeable menstruum, as in the following recipe :

R_x. *Oleoresinæ aspidii* 3j.
Tinct. quillaia f3ss.
Spts. aurantii dulcis f3j.
Syr. aurantii q. s. ad f3vij.—M.

Kousso appears to be used more by European than American physicians and is said to be efficient and free from danger. The freshly-prepared infusion is best used (*infusum brayeræ*, U. S. Ph.), but is very objectionable to children from its disagreeable taste, and is liable to produce vomiting.

Pumpkin-seed is a perfectly safe and simple remedy, but in my experience is never efficient; a small part of the worm being left behind to reproduce the trouble. The outer shell of the seeds should be removed, and the inside rubbed up with syrup or honey into an agreeable mass. One or two ounces of this can be eaten, followed, as in all cases, by a purgative.

Another agreeable remedy is the meat of the cocoa-nut. From large quantities of this there have been favorable reports lately, but as cocoa-nut is rather indigestible it might have an untoward effect on the child.

With pelletierine or male fern, preferably the former, properly given with all the details of treatment attended to, success should always finally crown our efforts, and it seems to me better not to waste time with any other remedies.

IV.—*TRICHOCEPHALUS DISPAR* (WHIP-WORM).

This is a small worm, thickened at one end, but tapering out like a whip-lash at the other. It is four or five centimetres long, and lives in the cæcum where it is often found in large numbers (Fig. 8). The eggs (Fig. 2, *d*) are about the size of the ova of the pin-worm, from which they are easily distinguished by the irregular rounded shape. At each extremity is a break in the egg-walls. Of 16 children examined for this purpose, I found the eggs of this worm in the fæces of one. The worm gives rise to no symptoms, as far as known.

FIG. 8.



Trichocephalus Dispar (Heller).

DISEASES OF THE LIVER.

BY JOHN H. MUSSER, M. D.,

PHILADELPHIA.

DISEASES OF THE LIVER are not of frequent occurrence in childhood. The factors essential for the development of hepatic disorder require the element of time to aid them. This is one reason gall-stones, for instance, do not occur in early life. Moreover, the customary food and drink of early childhood do not influence hepatic function and nutrition deleteriously, and therefore functional derangements, hepatic congestion, and sclerosis are relatively infrequent. Other etiological factors of liver disease in adult life are not operative in childhood. The liver is more frequently the seat of secondary disease than possibly any other organ. The primary diseases usually occur in adult life, and hence the secondary effects are only observed at that period. For instance, cancer of the liver and abscess following amœbic dysentery are not of frequent occurrence in childhood.

While the above applies chiefly to organic disease of the liver, the writer fully believes that functional disorder in late childhood and early adolescence is of more frequent occurrence than we are led to believe from the text-books. If the broad view of Murchison be true, that lithæmia and allied disorders with their long train of functional derangements in the gastro-intestinal tract, the nervous system, and the circulatory apparatus, or their results, terminate in organic disorder of liver, kidney, arteries or nerve-structure, we must believe that the beginnings are found in the errors of diet, the improper clothing, the misguided exercise, the vicious methods of education, and abnormal excitements of the nervous system which occur in childhood. It is true the physiological labors of the liver are so closely related to, or rather so markedly an adjunct to, the physiological labors of other organs of the primæ viæ that it is almost impossible to fix upon the disturbing factor when disorder is observed. Hence a clinical distinction between malnutrition and malassimilation cannot be made. Functional disorders, therefore, will not be discussed in this chapter, and for the above reasons are usually excluded in works on diseases of children.

Diseases of the gall-ducts, save catarrhal inflammation, are due either to gall-stones (not present in childhood) and their consequences or to diseases outside of the duct that do not arise in early life. Hence affections of these passages need not be considered.

General Etiology.—The causes of liver disease in childhood do not differ from those in adult life, though they are not as frequently operative, or the results of their operation are not seen. Icterus neonatorum and congenital obliteration of the ducts are the diseases of the liver peculiar to childhood, and therefore have a distinct etiology. Other affections of the liver are common to both periods. Errors of diet, excess in rich food or in stimulants,

cause congestion of the liver in children as in adults. Seasonal changes are factors, although it seems that high temperature does not often tend to cause acute congestion of the liver in children; at least, writers on tropical diseases do not specifically refer to the occurrence of acute congestion in early life. Malaria causes congestion of the liver at any age; checking of discharges or chronic constipation are not marked factors in childhood; but the congestions that arise in the course of infectious diseases are more commonly found at this time. Scarlet fever, measles, and, notably, relapsing and yellow fever, are attended by congestion of the liver. In passive congestion we find the same influences at work in the child and the adult. The effects of obstruction of heart and lungs are similar.

As in congestion, so in fatty liver, the causes are not peculiar. In children abnormally obese or the subjects of phthisis or profound anæmia, the disease is liable to occur, just as in adult life. The same is true of amyloid disease; prolonged suppuration alone or in tuberculous bone disease or in tuberculosis of the lungs leads to its frequent occurrence in children. Syphilis is a common associate and rachitis is occasionally observed with amyloid liver. Osler states that amyloid disease is found in prolonged convalescence.

Syphilitic inflammation of the liver in children is almost always congenital. In hydatid disease of the viscus we see a common cause at both periods of life, and as hydatids grow slowly, it is possible infection takes place in childhood, but symptoms do not arise until later in life.

In suppurative hepatitis the etiological factor differs at different ages. In this affection in children we do not find the baneful causative effects of high temperature, nor does it appear to follow amœbic dysentery as frequently as in adults. It is possible this form of dysentery is not common in children. The writer had occasion to analyze all the recorded cases of abscess of the liver up to 1890, and found that portal pyæmia and traumatism were more frequent causes than tropical dysentery (respectively 10 and 8 in 34 cases), and that round worms in the ducts were only slightly less common.

In cirrhosis of the liver, again, the causes are not dissimilar, although the infectious diseases play a more important part in childhood, while alcoholism is an infrequent causal agency. Klein points out the frequency of scarlatina, and Laure and Honorat, and Siredey, measles, as factors in its production. Tuberculosis is another cause. It is remarkable to find the affection occurring with general arterio-capillary fibrosis. Howard believed that rich, high-seasoned food is likely to produce cirrhosis in childhood, and that we have reason to believe ptomaines are causal agencies.

But little attention need be paid to the morbid anatomy and pathology of diseases of the liver in childhood. The morbid processes do not differ from similar processes in adults, and, as the scope of this article is limited, the discussion of morbid anatomy and pathological histology will be omitted.

General Symptomatology.—The subjective and objective symptoms of hepatic disease in childhood usually present the same striking picture of morbid change as in adult life. Apart from the symptoms that attend failing health, the subjective sensations of hepatic disorder are few. If we consider functional derangements of the liver to be the primary cause of lithæmia, then indeed the above remark is not correct; but, as previously noted, such relationship will not be considered.

Pain is a subjective symptom found only in one or two of the disorders which are to be discussed in this article. It occurs in suppurative hepatitis, in syphilitic inflammation of the liver when the capsule is involved, and in a slight degree in congestion. It may be localized to a small area, or

the whole organ may be the seat of pain. It is constant, increased by pressure or movement. It may extend to the right shoulder. The patient may be compelled to lie on the affected side with the legs drawn up. The paroxysmal pain that characterizes hepatic colic, and is the most frequent pain of hepatic disease in adults, does not occur. Pain in the region of the liver in childhood must be distinguished from pleurodynia and pleurisy. In pleurodynia there is immobility, respiration and other movements are painful, the area is tender on superficial examination by palpation, and other portions of the body may be affected with rheumatism, or there is a distinct history of exposure. In pleurisy the pain is markedly increased by breathing, is associated with a pleural friction, and is sharp and lancinating, attended by cough and increased by it. Pressure at a localized area increases it. It is often difficult, indeed impossible, to distinguish right-sided pleurisy from a perihepatitis. In both friction occurs; in the former, fluid may soon be detected in the pleural cavity, or the development of pneumonia may aid to distinguish the two. It may be said that the pain that attends liver disease is increased by pressure at any part of the liver, particularly upward along the lower edge of the viscus, or in the epigastrium.

Pain in simple abscess of the liver is localized; the locality corresponding to the seat of injury when that is the cause of the abscess. In pyelphlebitis the pain is more diffused. In abscess there is localized tenderness; in perihepatitis the parts are exquisitely tender on palpation. Weight and fulness and uneasy sensations are described by the patient when there is enlargement of the liver. They are not of diagnostic value.

The subjective symptoms referable to gastro-intestinal derangement are many, but are not characteristic. Loss of appetite, a bitter taste, nausea, dyspeptic symptoms, particularly flatulency, with irregular or costive bowels, occur. The objective symptoms—noted by the usual methods of physical examination—are jaundice, ascites, enlargement of the spleen and of the abdominal veins, hæmorrhoids, and fever and sweats.

Physical Examination.—The liver in infancy and childhood is larger in proportion to the weight of the body than in adult life. It therefore presents a relatively greater surface for examination. The left lobe is particularly accessible to physical examination. The upper border of the liver extends to the fifth, sixth, and seventh ribs in the mid-clavicular, axillary, and scapular lines respectively. The lower border extends two inches below the margin of the ribs. In the median line the left lobe extends to within an inch of the umbilicus.

Inspection.—The decubitus of the patient is not peculiar in hepatic affections except when acute inflammation is present. The recumbent posture is assumed and the legs drawn up. The patient may lie on the right side. If pain be present, it is increased by keeping on the left side. The abdomen is usually distended by flatus, or in certain affections by ascites. If the liver be enlarged, the right lower third of the thorax is distended, as well as the contiguous portion of the abdomen. If there is much enlargement or if acute pain is present, the movement of the right lower half of the thorax is limited. The epigastrium is distended. The swelling of the hepatic area may correspond to the entire organ or may be localized. In abscess and hydatid disease tumors may be detected in the left lobe of the liver, along the lower border of the right lobe, or as swelling with projection of the ribs at points corresponding to the convex surface of the liver. Hence the epigastrium, the right hypochondrium, and right lumbar region, and the mid-clavicular, mid-axillary, and scapular lines along the upper border, are the favorite seats of election

of tumors. In abscess the superimposed skin may be reddened. The appearance of the veins over the surface must be noted.

Palpation.—By palpation the position of the lower border of the liver and the character of its surface are determined. The former is easily ascertained if the abdomen is not too much distended and if the child can be kept quiet during the examination. The normally large left lobe must not be mistaken for a tumor. The liver moves with respiration, and this fact must be ascertained in order to exclude the presence of tumors in the abdomen due to other causes. Fæces in the transverse colon must be excluded by the administration of purgatives. The surface of the liver, as well as its edge, may be soft, as in fatty liver, or indurated, as in amyloid disease. In both the edge is smooth; in cirrhosis it may be sharp, but is invariably hard. Bosses may be detected due to cancer, hydatid disease, or abscess. In hydatid disease they are soft and may fluctuate; in abscess they are hard at first, then become soft and fluctuating. A friction vibration is sometimes detected by the palpating hand in cases of perihepatitis, and the peculiar fremitus may be elicited in hydatid disease. Oedema of the surface is observed occasionally in abscess.

Percussion.—By this means the size of the liver, whether diminished or enlarged, can be accurately determined, and the degree of enlargement ascertained. Marked deviations from the normal boundaries of percussion, as indicated above, serve to distinguish the changes. It must not be forgotten that to define the upper border deep percussion must be employed, and, to define the lower border, light percussion. The colon must be emptied of fæces, and the character of the evacuations noted. Affections of the pleura, particularly effusions, must be excluded. When a pleural effusion is present there is a uniform bulging of the side, the respiratory movement of the liver is restricted, and a depression is sometimes seen between the effusion and the liver if that organ be pushed down. By percussion it is found that the dulness of effusion is movable, and that its upper limit is S-shaped or horizontal. The rational symptoms of pleurisy aid to distinguish it. When the liver is enlarged the ribs are everted.

In determining the outline of the liver by percussion it is well to ascertain if it be regular or not. When the liver is enlarged in its entirety the normal shape is not departed from. If the enlargement is due to hydatid disease or abscess, the outline is irregular. The area of dulness may extend out from the normal liver in positions indicated by palpation. Sometimes the enlargement, though uniform, occurs in one direction only; thus in abscess or hydatid disease of the convexity the increase in dulness is upward and to the right; in hydatid disease of the centre, downward. Both affections may be limited to the left lobe, and then an increase in size of the corresponding area is noted.

Diagnosis.—By means of physical examination, with a study of rational symptoms, simulated enlargement of the liver is excluded. Apparent increase in the size of the liver, as determined by palpation and percussion, may depend upon congenital change in the shape of the liver or upon displacement of this organ by the deformities of the chest, due to rickets or to caries of the vertebræ. Congenital change in shape is recognized by the fact that it is noted soon after birth, and that, while it is persistent, symptoms of hepatic disease are absent.

Apparent enlargement of the liver upward—dulness extending to the fourth rib in front—may be due to tumors in the abdomen or to ascites; or the normal liver dulness may be continuous with the dulness due to sarcoma of the kidney, to tuberculous disease of the omentum, to an ovarian tumor, or to encysted or free fluid in the peritoneal cavity.

If fluid be present, the dulness may change if the patient turns on the left side; the lower border can then be defined. If the fluid be encysted, diagnosis is more difficult. A history of previous peritonitis or a history of tuberculosis, with associated development of the disease in other organs, with fever and emaciation, is suggestive of tuberculosis, which is usually the cause of encysted fluid as well as omental disease. A tumor of the right kidney may be distinguished from an enlarged liver if the tumor be rounded, if the fingers can slip between the tumor and the liver, if a tympanitic note, indicating the presence of the intestine, be found to run across the surface of the kidney, if the tumor do not move with respiration, and finally by urinalysis.

The physical examination is not complete unless the characteristics of the organs adjacent to the liver are observed. Without such examination no diagnosis can be made nor rational treatment conducted.

Operative exploration of the liver, accomplished by means of the aspirator or hypodermic syringe, properly sterilized, is useful to confirm the diagnosis of hydatid disease or of abscess of the liver. By this means three kinds of liquid may be withdrawn—serum, pus, or hydatid fluid. The former, serum, does not occur in the liver; either the pleura or the space underneath the diaphragm yields it; but its presence does not exclude hepatic disease, for serous inflammation may complicate the liver affection. Cases are recorded in which, after emptying the pleura of serum, deeper exploration through the diaphragm yielded pus. The association of pleurisy or empyema and subdiaphragmatic abscess with hepatic disease must not be forgotten.

By the aspirator clear laudable pus may be withdrawn. It often contains crystals of leucin and tyrosin, and, it is said, the characteristic liver cells. If such cells can be recognized, it is proof positive that the pus was originally in the liver. The pus may be so mixed with blood as to appear reddish-brown, like anchovy sauce. In this case, on microscopical examination, the *amoeba dysenterica* is sometimes found in the purulent fluid. The abscess is then secondary to dysentery.

Hydatid fluid is clear, alkaline, of low specific gravity, contains sugar, a trace of albumin, and a large amount of chloride of sodium. Succinic acid has also been detected. On microscopic examination hooklets, echinococcus membrane, sometimes scolices, and often hæmatoidin crystals are found. It is to be remembered that hydatid cysts may suppurate; pus will then be secured by aspiration, in which the remains of the echinococcus cyst are present.

In diseases of the liver in childhood an accurate diagnosis can be made only by a consideration of the personal history of the patient, of the previous diseases from which he suffered, of the evolution of the disease the nature of which is to be solved, the subjective symptoms and physical signs of the ailment, and the condition of all the organs and structures of the body. A systematic pursuit for all the facts, as embraced above, is necessary in the study of disease of any portion of the body; but the liver, more than other organs, is subjected to onslaughts of morbid action that primarily develop elsewhere; hence previous ailments must be investigated and the integrity of all the tissues carefully ascertained. For the differential diagnosis of the various affections this is essential. Of the hepatic affections discussed in this work, congenital disease of the gall-ducts, some forms of congestion, and hydatid disease are the only ones that are not secondary to affections of other organs.

A diagnosis is facilitated not only by inquiring into the integrity of the various organs of the body, but also by securing definite information regarding the occupation, habits, residence, and all other conditions of life of the patient. Illustrations could be advanced in any disease, but it suffices to

point out the value of the knowledge of alcoholism in cirrhosis, of exposure to phosphorus in yellow atrophy, of residence among dogs in hydatid disease.

JAUNDICE.

Etiology.—As seen most frequently in childhood, jaundice is due to obstruction of the bile-ducts—the *hepatogenous* form—resulting from pressure upon the ducts, or obstruction within them.

Pressure upon the Ducts.—Organic disease of structures adjacent to the ducts which might press upon them is very rare in childhood.

Obstruction within the Ducts.—Affections of the mucous membrane are abnormal processes very liable to occur in infancy. When the lining membrane of the ducts, and particularly the portion of the common duct known as the *pars intestinalis*, is the seat of catarrh, the membrane swells and causes obliteration of the lumen. Jaundice therefore occurs. Congenital obliteration of the ducts is also found to be a cause of jaundice. Gall-stones do not occur in childhood, and the wandering of worms into the duct is rare. It is seen, therefore, that the obstructive or hepatogenous form of jaundice is due in the larger proportion of cases to catarrh of the ducts and sometimes to obstruction of them by round-worms.

The causes of *hæmatogenous* or *non-obstructive* jaundice are also few in number. Yellow fever, malaria, epidemic jaundice, and pyæmia may be possible causal factors; poisoning by phosphorus, the use of ether or chloroform, mercurial poisoning, and snake-bite are rare possibilities. No cases of acute yellow atrophy in childhood have been reported.

Jaundice is a symptom, not a disease. It is recognized by symptoms and general physical signs.

Symptoms.—Icterus, or the yellow hue of skin in jaundice, is usually first noticed by the nurse or mother. The color varies from lemon-yellow to olive-green or a bronzed hue. In obliteration of the ducts it is most intense. It develops gradually, usually on the face first. In the obstructive form it is general. The conjunctivæ are deeply colored; the mucous membranes are tinted; the secretions are bile-tinged; the sweat stains the linen yellow. The urine is loaded with bile-pigment. It is brownish-yellow or has a greenish tinge. When shaken in a test-tube a yellow froth rises to the surface. By the nitrous acid test the play of colors characteristic of reaction with bile-pigment is seen. While the tissues and secretions are bile-tinged, the fæces are deprived of the pigment. They are pale or slate-gray in color, very offensive and pasty. The temperature is frequently subnormal. Prostration occurs, and anæmia arises. The influence of the bile on the nerve-centres or their peripheral terminations is seen in the character of the pulse, the occurrence of itching, and the grave cerebral phenomena to which the term *cholesteræmia* has been applied. The pulse-rate is much diminished; it often falls to two-thirds or one-half of the customary frequency. Itching is a most distressing symptom and is caused by the bile-pigment irritating the peripheral cutaneous nerve-filaments. Often the body, particularly the trunk, is covered with scratch-marks. The skin is liable to eruptions, as erythema and boils.

Ordinary cases of jaundice frequently show some irritability of temper and mental depression. This may be followed by drowsiness and by stupor ending in coma. In children convulsions are frequently seen. In malignant cases the typhoid state usually closes the scene; the pulse becomes rapid, fever occurs, the tongue is dry and brown, sordes collect on the teeth, and there is *sub-sultus tendinum* with low delirium, and sooner or later convulsions and coma.

Here too hæmorrhages occur, the leakage being subcutaneous or into the mucous membranes, and appearing as nose-bleed, hæmatemesis, or melæna.

Epidemic jaundice occurs at times in children. Denton reports a small epidemic among children of the same school. The symptoms were sudden vomiting, headache, vague gastric pains, with prostration, and in three or four days intense jaundice. Duration, ten to twelve days. Hennig, after a study of three house-epidemics of infectious icterus, concludes that it is a general acute, specific, infectious, miasmatic, non-contagious disease. It may be sporadic, epidemic or endemic, and appears to have a relation to typhoid fever and to typhus. The infectious agent arises outside of the human body. The disease runs a favorable course and never relapses.

Raven believes ordinary catarrhal jaundice may be infectious, and reports an instance in which one child of a family became icteric from exposure, and that four others of the house developed the affection, apparently by contagion.

Diagnosis.—The diagnosis of jaundice is not difficult. The greenish-yellow hue of chlorosis, with the pearly conjunctivæ, would suggest an examination of the blood, the result of which would distinguish chlorosis and jaundice. Similar examination would enable an exact diagnosis of pernicious (idiopathic) anæmia to be made in cases resembling jaundice in the straw-colored skin and the conjunctivæ made yellow by the deposition of fat. The rarity of Addison's disease in childhood is such as to preclude the possibility of an error in diagnosis. The same may be said of malignant disease of the abdominal viscera. Malaria, however, occurs at any age; but the rational symptoms, the plasmodia and pigment in the blood, and the condition of the spleen aid in the diagnosis of the paludal disorder.

Varieties of Jaundice.—**JAUNDICE IN THE NEW-BORN.**—In the new-born infant jaundice occurs in mild form during the first week of life on account of ligation of the cord and consequent alteration of blood-pressure in the liver, and in malignant form in (1) congenital obliteration of the biliary passages, and (2) pylephlebitis secondary to inflammation of the umbilical vein.

Simple jaundice in infants rarely produces grave symptoms. The skin, the conjunctiva, and the mucous membranes show a yellow discoloration, varying in degree in different cases. The urine is loaded with bile-pigment. The child sleeps more than in health, and may not arouse when feeding should take place. The bowel movements may be pasty and white. Such jaundice begins twelve or twenty-four hours after birth. It lasts two days to a fortnight. The infant usually remains well nourished. It is due to low tension in the blood-vessels of the portal circulation (after ligation of the cord), which causes rapid absorption to take place from the bile-capillaries in which the tension is higher. Quincke thinks it is due to patency of the ductus venosus.

Icterus neonatorum is to be distinguished from the pseudo-jaundice that occurs after birth due to a destruction of red corpuscles in excess of the powers of the liver to discharge them from the body in the bile. In this condition the conjunctiva is not injected, the stools are not clay-colored, and the urine does not contain much pigment. The discoloration fades like a bruise from yellowish red to flesh color. It is said late ligation of the cord allows a portion (one-half) of the blood in the placenta to flow into the infant's body, and therefore this distends the foetal vessels by so much. This fact is of importance if, as Parks states, distended blood-vessels exhibit more intense jaundice.

The treatment of the mild jaundice of infants is very simple. The bowels should be opened by a mild laxative, such as calomel or gray powder in minute

doses, or a few grains of calcined magnesia. The kidneys should be kept active by nitre or citrate of potassium well diluted. The child should be aroused to be fed, and the effects of the jaundice on the nerve-centres should be carefully watched. Ammonia in the form of the muriate or the aromatic spirits should be given, as in the following prescription :

R \bar{y} . Ammon. chloridi gr. j.
Syr. acaciæ f $\bar{3}$ ss.—M.

Sig. A coffee-spoonful every two hours.

Or,

R \bar{y} . Spt. ammon. aromat. f $\bar{3}$ j.
Syrupi f $\bar{3}$ vij.—M.

Sig. One-half teaspoonful every two hours.

Spirits in the form of whiskey in hot water may be given if there be depression. Hot water, sweetened, can be given with advantage in copious drafts, particularly when fasting, for its effect on the liver and kidneys.

There does not seem to be any reason against the use of gentle massage and faradism; both are vaunted in catarrhal jaundice in later life. Externally mild sinapisms, with light friction, must be employed if the circulation fails; and the extremities must be kept warm.

Jaundice due to Congenital Obliteration of the Bile-passages.—Four forms of obliteration have been noted: First, that in which no passage exists between the liver and duodenum; second, in which there is one permeable canal, but no exit from the gall-bladder; third, in which both cystic and hepatic ducts are obliterated; and, fourth, in which obliteration has taken place below the junction of the cystic and hepatic ducts.

Congenital malformation, with narrowing of the lumen of the parts on account of defective development, may exist to such degree that it leads to sluggish discharge of bile, which causes irritation of the ducts. A catarrhal process is set up, and leads to complete obliteration. The process is slow, but the obliteration is finished in some cases during intra-uterine life; in others not until a few months after birth. In a few cases the inflammation of the ducts and the surrounding parts has led to localized peritonitis. In all cases, "biliary" cirrhosis of the liver has developed secondarily.

The condition is rare. Dr. John Thomson was able to collect 64 cases. We are indebted to his monograph for the following facts: The parents of the children affected with obliteration of the bile-ducts are usually healthy. Syphilis in the parents is not an important factor. In several instances more than one child of the same family was affected, and in a large number of instances nearly all the children of families in which one case occurred had infantile jaundice or were subject to digestive disturbances. The character of the labor did not seem to influence the occurrence of the disease. At birth the affected child presented no abnormal appearance, except jaundice. In 2 out of 60 cases the lesions of congenital syphilis were seen. Boys were affected more frequently than girls.

Jaundice is the most pronounced symptom. It is most frequently present at birth, but may not develop until one, two, or six days after, and may be delayed beyond a fortnight. It soon becomes of a greenish hue, and it progressively deepens until the final termination. The urine contains bile coloring matter. The meconium may be normal or colorless. When it is normal the obliteration has taken place late in uterine life or not until after birth. The motions are whitish-gray at first or become so immediately after

the meconium is passed. At times green matter is voided with the stools. It may be due to mercury which had been administered or to micro-organisms in the fæces.

Next to jaundice, the occurrence of spontaneous hæmorrhages is the most frequent and characteristic symptom. They occur subcutaneously, from the umbilicus, the bowels, the stomach, the nose, and other portions of the body. The occurrence of hæmorrhage is of very bad prognostic omen, death usually occurring a short time afterward. Usually in jaundice the blood-corpuscles are so reduced as to create the hæmorrhagic tendency, but Thomson believes hæmorrhages occur because of some change in the blood-vessels produced by an excess of ptomaines in the blood, the function of the liver by which these poisonous materials are destroyed being in abeyance.

With or without hæmorrhage, convulsions frequently take place. These phenomena are of frequent occurrence in other forms of jaundice, and are not peculiar to the affection under consideration.

Progressive and easily recognized enlargement of the liver and spleen takes place, with the development of the grave phenomena indicated. Emaciation and exhaustion rapidly progress, and death ensues from slight intercurrent disease, from coma or from exhaustion.

The diagnosis is not difficult; the prognosis of a fatal termination is positive. The duration is from one week to four months. Two cases recorded by Thomson lived to the eighth month. Treatment is without curative results.

Jaundice due to Inflammation of Umbilical Vein.—Icterus may occur in infants because of inflammation of the umbilical vein, with secondary pylephlebitis. The stump of the cord is swollen and may exude pus, or the navel is ulcerated and inflamed. Hæmorrhage is likely to arise. The skin is discolored around the navel, and the parts are tender. The liver is enlarged, and may be tender over the surface. In rare cases a localized or general peritonitis occurs. The attack may be ushered in with a convulsion, which is apt to recur. The infant is restless and cries very much. The desire to nurse is lost. Vomiting occurs, and often diarrhœa soon sets in. Foci of infection arise in other structures—the joints, the brain, the lungs. The joints become painful on movement and are swollen and red.

After the convulsion, or perhaps without it, fever sets in with the customary phenomena. The temperature is high and may be intermitting; the pulse is very rapid, the respiration increased; cough may be present; jaundice is not very intense. As the temperature rises the liability to convulsions increases, and death follows the convulsions, occurs in coma, or may take place from exhaustion. After death a septic pleurisy, pericarditis, peritonitis, or meningitis may be found, or similar inflammation of the kidneys observed.

The fever, the local signs and symptoms, and the jaundice render the diagnosis easy. In a few cases the local signs are not noted, under which circumstances the difficulties are greater. The prognosis is most grave. The treatment is simply symptomatic. Prevention of this fatal illness of the new-born must be sought in strict antiseptic dressings of the cord. Often a cord bleeds after the first ligature. The second tying is most dangerous unless done with proper precautions. The writer had a case of this character in which infection took place from and at the hands of a dirty nurse. Before ligating the cord dirty rags were applied to attempt to control the hæmorrhage.

Jaundice in Winckel's Disease.—Jaundice is seen in that fatal affection of the new-born known as Winckel's disease, or acute hæmoglobinuria. Cyanosis and hæmorrhage occur with the hæmoglobinuria, but the liver and spleen do not enlarge.

JAUNDICE IN LATER INFANCY AND CHILDHOOD.—Icterus occurs at any period of childhood and in both sexes. It is usually of the so-called catarrhal form. Errors of diet, improper food, excesses, irregular meals, improper clothing, exposure and chilling of the extremities, leading first to gastro-intestinal catarrh, are common causes.

The onset is gradual, being preceded by the symptoms of acute or subacute catarrh of the stomach and duodenum. There is some tenderness in the epigastrium and the right hypochondriac region, the liver is enlarged and may extend an inch or two below the normal line, and the characteristic signs and symptoms of jaundice are present. The hue does not change to the green or bronzed yellow of malignant jaundice. Hæmorrhages do not often occur. A moderate degree of fever is observed for a short time. The course may extend over three or four months.

The diagnosis is not generally difficult. A history of long-continued improper feeding or of a sudden attack of vomiting, etc. from improper food or from cold, is usually elicited. The gradual development of the jaundice, with relatively slight constitutional symptoms, with moderate fever only, aids in the recognition of the character of the affection. The causal presence of worms or hydatid cysts in the ducts cannot be distinguished during life.

The prognosis is good.

Treatment.—If fever be present, rest in bed must be enjoined. The extremities must be kept warm. Mild counter-irritation over the epigastrium, by means of sinapism or frictions with stimulating liniments, may be employed; massage is also beneficial. Gerhard advises compression of the gall-bladder or gentle manipulation in that region. Faradism has also been advised. The diet must be bland and free from saccharine or amylaceous articles. Milk diluted with an alkaline or carbonated water or with lime-water and taken hot, koumyss if vomiting be present, junket, and animal broths, such as beef-tea, mutton-broth, and chicken-tea, may be administered. After the acute symptoms have subsided semi-solids may be used. Preparations of milk and eggs, beef-jellies, oyster-broth, and clam-broth are appetizing. Light fish may be selected as convalescence proceeds, and sweet-breads, broiled beefsteak, and the white meat of chicken.

If there be much gastric disturbance, sedatives must be used. Calomel in small doses, calomel and bismuth, effervescing alkaline waters, carbonic-acid water, citrate of potassium in officinal solution favorably made, are of service. If there be pain, minute doses of magnesia may be added to the mercurial powder, or paregoric may be given with the citrate of potassium:

R_x. Liq. potassii citratis f̄3ij.
Tr. opii camph. f̄3j.

Sig. One-half to one teaspoonful every two or three hours.

Hydrochlorate of cocaine in solution sometimes allays the vomiting. If there be constipation, an enema sufficient thoroughly to evacuate the bowels frequently relieves the vomiting. Afterward, if necessity requires, the bowels should be opened by a mercurial, as calomel or gray powder, in small, frequently-repeated doses, or by the citrate of magnesium, or a saline purgative, as Hunyadi, Friedrichshall, Bedford, or Saratoga water.

When the acute symptoms are ameliorated, it remains to treat the catarrhal inflammation of the duodenum and ducts and the symptoms due to the jaundice.

In the treatment of catarrh the diet, as indicated above, must be persisted in; small doses of bismuth may be continued. Nitrate of silver in small dose,

with opium if pain be present, is a valuable sedative which modifies the catarrhal process. In young children it may be given in solution and should be administered on an empty stomach :

R_x. Argent. nitrat. gr. ss.
Mucilag. acaciæ f̄ij.—M.
Sig. Teaspoonful three times daily to a child under two years.

Oxide of zinc, in doses of one-twelfth of a grain every three hours, is also useful.

Small doses of ipecacuanha are often, after acute symptoms have subsided, of service. One-fourth to one grain of the powder three times daily is praised highly by many.

Phosphate of sodium is a most valuable drug in catarrhal jaundice. Ten grains three times a day in milk for an infant or half-drachm to one drachm for a child of ten, in hot water, and taken fasting, proves of inestimable benefit. It may be used with other remedies.

Chloride of ammonium is much used, particularly in India; one to five grains of the drug every three hours is frequently followed by surprising results. It may be administered in syrup of licorice or in syrup of orange. It does appear to dissolve toughened mucus, to allay congestion, and to promote secretion from the glands in the tubes.

Pilocarpine in doses of one-sixteenth of a grain has been recommended. It seems to have been of great benefit to adults.

After the tongue cleans, or, as is often the case, its epithelium is restored and the papillæ assume a normal aspect, the sedative remedies may be discontinued and a weak bitter or an acidulated bitter may be given :

R_x. Acid. hydrochlorici dil. ℥xxxij.
Infus. serpentariæ f̄ij.—M.
Sig. Teaspoonful in water before meals.

For more chronic cases dilute nitric acid internally and the local pack of nitric acid are often serviceable.

If the jaundice be of malarial or gouty origin, quinine in the former, or colchicine in the latter, has been often prescribed.

Finally, to treat the catarrhal process, the method of Krull is strongly insisted upon: Two to four pints of water are injected into the colon two or three times daily. The temperature is raised at each enema. The first enema is given with the water at a temperature of 59° F. It is made two or three degrees warmer until enemata at temperature of 72° are given. Krull and others testify warmly to its beneficial effects in children. The writer has seen most surprising results in adults, and, as no harm can result from its use, would not hesitate to use it in children.

Of the symptoms of jaundice requiring especial attention, itching may be mentioned. Sponging with sedative lotions is of service. Ten drops of carbolic acid to a pint of water, a solution of the bichloride of mercury, 1 to 3000, hot solutions of alkalies, as bicarbonate of sodium or borax, a drachm of each to the pint, may be employed.

Pilocarpine is recommended by Goodhart. He preferred to give it hypodermatically; $\frac{1}{24}$ to $\frac{1}{32}$ of a grain should be given to children over four years old. Since it was advised by Goodhart a number of physicians have commended its use. Internal diaphoretics of domestic origin at times are of

service. An infusion of sage or hot drinks, with a stimulant, excite perspiration and relieve the itching.

Intestinal dyspepsia with flatulency and painful digestion require some medication. The diet should in a measure prevent the development of these symptoms; nevertheless, they occur. Preparations of pancreatin given an hour after meals, with an alkali, will aid much in digestion. If they are not of service, such drugs as correct fermentation in the intestines must be administered. Of these, salol, naphthalin, and thymol are of great service, while creasote, carbolic acid, and charcoal may be given with advantage. Salol may be administered in powder or compressed pill. Naphthalin and beta-naphthol should be given in gelatin-coated pill or capsule. The coating does not dissolve until the drug reaches the intestine, and hence is of great advantage. Creasote or carbolic acid may also be given in pill or in emulsion with syrup of acacia. A prescription like the following generally overcomes the disagreeable symptoms:

R_y. Creasoti gr. $\frac{1}{4}$.
 Carbonis lig. gr. j.
 Pancreatin gr. j.
 Bismuthi subnitrat. gr. iij.—M.

Ft. chart. No. i.

Sig. Take after meals.

Or,

R_y. Acidi carbolic gtt. iv.
 Sodii bicarb. ℥j.
 Spiritus chloroformi f℥ij.
 Pulv. acaciæ
 Sacchari albi āā gr. xx.
 Aquæ q. s. ad f℥ij.—M.

Sig. A teaspoonful after meals or every three hours.

In selecting creasote the drug made from the beechwood must be used, and willow charcoal is preferable to the animal form.

The cerebral symptoms of jaundice can only be overcome by hastening the elimination of bile and at the same time supporting the patient. Stimulants must be used; preparations of ammonia, alcohol, and caffeine are to be selected. The preparations of ammonia are probably the best. Of course the patient must be nourished, and, if necessary, caffeine and cocaine may be resorted to. Both are advantageous stimulants, because they cause increased secretion from the kidneys, which are chiefly concerned in eliminating the bile. The poison without doubt sets up nephritis. It is necessary to guard against this complication if possible. Creating diaphoresis by jaborandi or the hot vapor-bath brings about this result. The kidneys may be relieved also by local applications, and particularly by the use of dry cups. In the case of more or less persistent jaundice these organs should be relieved quite frequently in the manner just suggested. The alkaline waters that may be selected for their beneficial effects upon the liver should also have diuretic properties. If they are not sufficient, the citrate of potassium or cream of tartar lemonade may be given.

The slow pulse, the subnormal temperature, and the prostration that ensues in jaundice are to be treated in accordance with the general principles of the management of these conditions. If hæmorrhages occur, turpentine or erigeron may be administered internally. Sulphuric acid and the acetate of lead are

also valuable astringents. The blood is always reduced in jaundice, the red corpuscles diminished in number. It is possible the systematic inhalation of oxygen may prevent this diminution, or at least combat symptoms depending upon it. It certainly is worthy of trial.

CONGESTION OF THE LIVER.

Both the *active* and *passive* forms are seen. *Active congestion* is acute, and is induced by an exaggeration of all circumstances which increase the physiological congestion that takes place under the stimulus of food. Over-eating, the eating of rich food, the abuse of stimulants, are liable to cause an acute attack of hepatic congestion. Excess of heat may superinduce an attack in hot climates.

The symptoms are much like those of catarrhal jaundice, with the physical signs of enlargement of the liver. The jaundice is not intense. The face becomes sallow and cachectic if jaundice be absent. The patient loses in health and strength. Some pain is complained of in the hepatic region, which is tender on palpation. The liver is enlarged uniformly in all directions, often extending two inches beyond the normal boundaries; the edge can be felt and is smooth and rounded; the surfaces also are smooth. In a few cases the gall-bladder is enlarged, and can be detected in the right hypochondriac region to the left of the midclavicular line in a line drawn from the acromion process of the right shoulder to the umbilicus.

With the removal of the cause the symptoms disappear, and by the end of a month the functions of the gastro-intestinal tract are restored and the liver is reduced in size. In some cases enlargement of the organ and the peculiar complexion of the patient continue for a longer period.

Passive Congestion.—The passive form of congestion is associated with disease of the heart and lungs and chronic malarial poisoning. The pronounced symptoms are due to the disturbance of these organs; along with congestion in other organs the liver becomes engorged with blood, and hence gradually enlarges. The shape of the enlargement is similar to that in active congestion. The edge of the liver is likely to be sharper and more indurated. No nodules can be detected on the surface. In the right midclavicular line the lower border may extend to the level of the umbilicus, and in the median line the left lobe may extend three-fourths the distance. Frequently the upper border cannot be so readily made out, because of the occurrence of effusion into the right pleura. The rational symptoms are those of mild gastro-intestinal catarrh. The tongue is furred; there are nausea, loss of appetite, and intestinal dyspepsia; vomiting and constipation may occur, or there may be diarrhoea. A slight form of jaundice is developed. Albuminuria is observed, and the urine presents the appearance of congestion of the kidneys. On account of the interest centred in the condition of the heart and lungs passive congestion of the liver is frequently overlooked.

Diagnosis.—The diagnosis of active and passive congestion of the liver is made without difficulty. The presence of a cause for the congestion, together with the mode of onset, are pronounced factors in the diagnosis.

Prognosis.—In the acute forms the prognosis is generally favorable. In chronic congestion the prognosis is modified by the knowledge of the cause of the congestion.

Treatment.—The removal of the cause is essential to the successful management of active congestion of the liver. Correction of errors in diet, in habits of life, or in occupation often suffices to relieve the affection. The gastro-

intestinal symptoms are treated as in catarrhal jaundice. More stress must be laid on the use of purgatives for depletion. The alkaline waters and the mercurials are of benefit. Phosphate of sodium is useful; it may be given in hot solution on an empty stomach either at night or on rising in the morning. The hygienic and dietetic management employed in catarrhal jaundice is of use in active congestion of the liver. In hot climates, if such congestion occur, two drugs are used and lauded. The chloride of ammonium in 3- to 5-grain doses, every two or three hours, relieves the discomfort and appears to remove the engorgement of the organ. Ipecacuanha is used for a similar purpose. The drug must be given in large doses, administered twice in the twenty-four hours; 5 grains to children under five years of age is admissible. In order that vomiting should not be caused by the drug, the administration should be preceded by a few drops of the deodorized tincture of opium and a sinapism applied to the epigastrium. Twenty minutes after the application the drug may be given. After the more acute symptoms have subsided bitter tonics should be prescribed. If, however, there is pronounced gastric catarrh, small doses of calomel or bismuth or nitrate of silver, as advised in catarrhal jaundice, may be administered. One of the mineral acids, especially dilute nitric acid, in small doses, is given after the subsidence of the acute symptoms, particularly if the liver does not diminish in size.

Passive congestion of the liver is treated by alleviating the symptoms due to the engorgement, and by the employment of measures and remedies to relieve the primary cause of the disease.

FATTY LIVER.

Enlargement of the liver due to fatty infiltration or degeneration occurs in the course of other diseases or on account of improper habits of the patient. In children it is always an intercurrent affection. Tuberculosis and wasting diseases generally are associated with fatty infiltration. The wasting that attends gastro-intestinal catarrh is associated with fatty liver. This is particularly the case if the catarrh results from the excessive use of sugar and starchy food. The enlargement is due to an accumulation of fat in the liver, and not to degeneration of the structures. It is said that children who are closely confined and have become anæmic are liable to this disease.

Symptoms.—The subjective symptoms are negative. Enlargement of the liver, which is uniform in all directions, is observed. The organ is of doughy consistency and the edge is rounded. The surface is smooth and painless on palpation. Jaundice, ascites, and other symptoms due to hepatic disorder do not occur.

Treatment.—The treatment depends upon the cause. If enlargement from fat accumulation is found in children who tend to be obese, and who have been indiscreet, strict hygienic and dietetic management must be invoked. The carbohydrates must be excluded from the diet; out-door exercise must be carefully planned, and if it cannot be indulged in, massage and Swedish movements must be directed. Sea-air has been advised in cases of this character.

AMYLOID DISEASE OF THE LIVER.

In this form of liver disease the organ is enlarged and but few hepatic symptoms of a subjective character are observed. The affection is associated with amyloid disease in the spleen, kidneys and intestines. The degeneration occurs in the course of phthisis, chronic bone disease, prolonged suppuration, and rickets. It may occur at any age throughout childhood.

Symptoms.—Anæmia is a prominent general symptom, and the pallor of the face is striking. The liver is enlarged in all directions; undue prominence of the abdomen in the course of any of the above-named affections should lead to an examination of this viscus. In addition to the enlargement of the liver, the spleen is also found to be enlarged. The liver sometimes attains a very large size; it may be twice or three times the normal weight. The surface is smooth, the edges hard and rounded. No pain attends palpation. The external veins may be distended; but jaundice does not occur, and ascites only results from diseases in other parts of the body, generally from the condition of the kidneys. Diarrhœa is usual, and hæmorrhage from the bowels may also take place.

Diagnosis.—The nature of enlarged liver occurring in the course of the diseases previously indicated can usually be determined without much difficulty. The diagnosis is rendered more positive by the detection of similar disease in the spleen and by the occurrence of albuminuria and polyuria due to amyloid disease of the kidney. The recognition of amyloid disease should be attempted in all cases in which operative measures for the relief of bone disease or suppuration is contemplated. Any grave operation will be contraindicated by the presence of this complication.

Treatment.—Notwithstanding the frequent suggestion by prominent authorities of the use of alkalies and the preparations of iodine in the treatment of this affection, there does not seem to be any drug which modifies or changes the course of the disease. The removal of the cause, if possible, is the most rational method of treatment. The few symptoms that are caused by the functional derangement or enlargement of the liver are to be treated. It must not be forgotten that in some cases it is almost impossible to say how much amyloid disease is present or to what extent the enlargement of the major organs within the abdomen is due to congestion. The symptoms and etiology may point with certainty to the presence of amyloid disease. If in such cases the heart be weak or there be organic disease, venous congestions of the viscera may also take place; and apparently hopeless amyloid disease may be cured by recognition of this pathological fact, and hence by resorting to removal of the cause by the administration of digitalis, strophanthus, and other heart-tonics.

SYPHILITIC INFLAMMATION OF THE LIVER.

The morbid process above indicated due to the special specific poison is seen in the congenital forms of the disease in childhood. Two forms of inflammation occur—one in which the disease is limited or in large part confined to the capsule; the second, in which the connective tissue of Glisson's capsule is the seat of inflammation.

Symptoms.—The symptoms are generally seen in children who have the characteristic appearance of face, trunk, and extremities of congenital syphilis, elsewhere described in this book. The skin eruptions, coryza and other mucous inflammations, anæmia, emaciation, and malnutrition, and, later in life, the appearance of the teeth, complexion, and shape of head, render the recognition of congenital syphilis comparatively easy. In perihepatitis there is much pain over the liver, breathing is difficult, and there is fever. The temperature rises to 100° or 101°, the pulse is frequent, the countenance distressed. Relief to the pain takes place when the patient assumes the upright position and crouches forward, or when he lies on his back with the legs drawn up. The marked tenderness interferes with palpation and

percussion. When the pain subsides the organ is found enlarged and the edges hard. After a week or ten days the more severe symptoms abate and convalescence is rapid unless the patient be broken down by previous bad health. Recurrence takes place on exposure or fatigue or without apparent cause.

In another group of cases the shrinking of new-made connective tissue begins, and soon the organ is grasped in the toils of fibroid overgrowth, contraction takes place, and all the symptoms of portal obstruction arise.

Jaundice may be the only manifestation of infantile hepatic syphilis. It is in all probability due to perihepatitis, with compression of the gall-duct, or to enlarged glands, which likewise compress it, or, most frequently, to adhesive inflammation of the portal vein.

Syphilis may be the cause of cirrhosis of the liver. The symptoms are twofold—one due to the congenital taint with possible associated lesions in other structures; the other, to portal obstruction. The latter symptoms do not differ from those of portal obstruction in cirrhosis of the liver of alcoholic origin.

Diagnosis.—The diagnosis of syphilitic disease of the liver is determined largely by the association of the lesions and well-known appearances of congenital syphilis, with symptoms indicating inflammation and functional disorder of the liver. Often the symptoms, and particularly the objective ones, are not obvious. The apparent alteration in size of the liver is not demonstrable; there is little if any pain, and features of portal obstruction are not observed. Jaundice may be the only symptom present. It is well to bear in mind that persistent jaundice in childhood without apparent cause, certainly if the gastrointestinal tract be free from catarrh, may be of syphilitic origin. The therapeutic test often aids in making a diagnosis.

Treatment.—The treatment is largely that of the cause, the remedies applied for the relief of congenital syphilis being indicated. In addition to the constitutional treatment the pain, jaundice, ascites, and other symptoms are to be relieved by methods previously indicated in this paper.

SUPPURATIVE HEPATITIS.

Two varieties are seen. In one the abscess is single, and in the other multiple; in the former the suppuration in nearly all the cases is secondary to trauma; in the latter suppurative pylephlebitis has occurred on account of suppuration in the portal area.

Symptoms.—The symptoms in the two forms differ entirely. In traumatic abscess, after the injury there is much pain in the hepatic region and symptoms of perihepatitis. The parts about the seat of injury are swollen, and the external surface may show the signs of a blow. After the injury the pain may diminish and the child be apparently well, when a recurrence of the local symptoms will arise; or the effects of the injury may not subside in the usual time. Pain in the region of the liver will be complained of, and on examination the organ is found to be enlarged. The enlargement is not uniform. It may be upward only, or, as is most frequently the case, be indicated by extension of the lower border of dulness downward. On palpation the hepatic region is painful; œdema over the most painful part or over the hepatic area or the area of enlargement may be observed. If the abscess be developing in the right or left lobe, an undue prominence may be seen in the right hypochondriac or in the epigastric regions respectively. It will be noted to move with respiration and to be continuous with liver dulness on percussion.

With the development of the local signs of enlargement and inflammation general symptoms arise. The fever, which may have been due to trauma, does not disappear, and indeed becomes more pronounced. It assumes a remittent or even distinctly intermittent type, and may be preceded by daily rigors and followed by exhaustive sweats; prostration ensues, and there may be a loss of flesh. The tongue is furred, appetite lost, vomiting may occur, and diarrhœa is frequently present. If the inflammation be seated on the convex surface of the liver, breathing is interfered with and cough may be present; both respiratory acts will in all probability be attended with pain. The pain is then noted in the sixth or seventh interspace in front or the seventh or eighth interspace behind. It may extend to the right shoulder, and in some cases pain in this position alone is complained of.

As previously intimated, sometimes the symptoms of suppuration, with local signs of inflammation, do not develop until a long time has elapsed after the injury. The general symptoms may arise before local signs of inflammation are evident. Between the injury and the development of the symptoms the child is not in good health. Loss of appetite, languor, inability to exert himself as was his former habit, with loss of flesh and strength, are very likely to be present.

Multiple abscess of the liver is usually preceded by a history of suppuration, and therefore a point of infection somewhere in the portal area. An appendicitis is one of the most frequent affections which precede this form of suppuration. It is thus seen that active abdominal symptoms may be present prior to the development of symptoms indicating involvement of the liver. If in the course of such symptoms jaundice arises and the liver becomes enlarged and painful, we may well suspect that the inflammation has spread to the portal vein. The type of the fever may also change. It becomes distinctly intermittent, and daily chills attend it. The onset of jaundice is characterized not only by the discoloration of the skin, but by the development of symptoms of the typhoid state. Delirium of a low muttering character soon occurs, deepening into stupor. The tongue becomes dry and brown, sordes collect about the teeth and lips, and subsultus is seen. In some instances convulsions occur; in others death takes place from exhaustion. Diarrhœa, if not previously present, is sure to arise. The stools are offensive and watery, and contain light-colored fecal matter. The urine contains bile-pigment, soon becomes scanty and high-colored, and is found to contain albumin and to have blood, epithelial, and granular casts. The nephritis may become so marked as to be a serious, indeed fatal, complication.

The patient usually lies on the right side, and when he assumes the opposite position complains of a heavy, dragging sensation. The skin is sallow, the complexion muddy. The facies is quite characteristic. Waring describes the appearance as follows: Countenance expressive of anxiety, shrunk, collapsed, pale, livid, or parchment-like.

Diagnosis.—If the symptoms of suppuration just indicated arise after trauma or the occurrence of suppuration of the portal area, diagnosis is not difficult. The cases of suppuration secondary to worms in the hepatic duct, or to suppurative inflammation of the ducts, extremely rare in childhood, are recognized with difficulty. The absence of a focus of suppuration in any other portion of the body when hectic symptoms are present should determine the necessity of careful examination of the liver. Enlargement, either general or local, may be made out by careful percussion. The exploratory needle may render positive a suspicion of hepatic suppuration, but the negative results of puncture do not exclude abscess. Friction-sound at the base of the right

lung, with diminished expansion of that side, may call attention to possible hepatic suppuration.

Reference has not been made to abscess of the liver occurring in the course of dysentery. The writer has not been able to find any recorded cases of this association in childhood, though there is no special reason why it should not occur. In cases of dysentery it is important to interrogate as to the condition of the liver, and, on the other hand, in acute liver affections the presence or absence of dysentery is to be ascertained. Amœbæ in the stools, in pus from an abscess, or in expectoration would confirm the diagnosis of this form of abscess of the liver.

Prognosis.—In multiple abscess of the liver the prognosis is very grave, such cases terminating fatally. In single abscess, if the pus can be reached by aspiration or by the knife, the prognosis is much more favorable. If the abscess be beneath the diaphragm in the upper portion of the right lobe, the issue is much more doubtful than when superficial.

Treatment.—The management of a case falls entirely into the hands of the surgeon. In multiple abscess of the liver no measures are of avail. In single abscess or where the number is limited to three, free incision must be made and may result favorably. If the abscess be situated along the margin of the ribs or in the epigastric region, the operation is simple and reparation takes place rapidly. The writer has seen two such cases recently in the Philadelphia Hospital. An abscess of the convexity of the right lobe must be reached through the pleural cavity. Excision of the ribs is necessary, and isolation of the pleural cavity quite essential. After pus is secured and the cavity drained and irrigated, a drainage-tube must be inserted and the case treated by the usual surgical methods. Recently the writer reported a case under his care in which Dr. Willard performed the operation above indicated most successfully.

HYDATID DISEASE.

This is a comparatively rare affection in this country. It seems, however, to be on the increase; within the last two years the writer has seen six cases, and knows it to have been more common in the experience of others. In children it is even more rare than in adults. With the exception of a child under twelve at the University clinic, no cases have come under the writer's observation. In the literature of the disease few if any cases are reported under two years of age. The liver is one of the organs most frequently affected. In childhood it appears to be the organ selected in 70 per cent. of the cases. In the recent exhaustive work of Graham a few cases only are recorded. He states that within a period of one year he observed hydatids in ten children, their ages varying from five to eight years. The youngest case that he refers to is one operated on by Thomas, a boy aged two years and one month. This disproves the statement of Leuckart, who at the time of his publication believed the youngest cases recorded to have been four and six years of age respectively.

There is, therefore, no immunity for children if their associations are such as to cause infection. The infection may occur very early in life, but the slow growth of the cyst makes it possible that they are not recognized for years. Moreover, in childhood, as Graham remarks, "the organs in which the cysts are situated are less likely to be so completely affected as is the case in the adult subject where the pressure changes are more permanent."

Space will not permit a discussion of the mode of development or infection of the human species. The growth in the child and the manner of its infec-

tion do not differ from the same in adults (for description of which recent text-books on pathology contain sufficient information).

Symptoms.—The cyst in the liver may develop and reach a large size without recognition. Attention is first called to its presence by the occurrence of mechanical symptoms; the abdomen enlarges or there is enlargement and swelling of the liver region. On examination, if the liver be the seat of the disease, it is found to be enlarged. The enlargement may be uniform; usually, however, it takes place in a particular direction. If the growth springs from the convex surface of the liver, the area of dullness extends higher in the axillary region and behind in the scapular line. If it begins in the right lobe, and the lower portion thereof particularly, the extent of dullness is increased downward toward the umbilicus. Sometimes the tumor is confined to the left lobe of the liver, and hence is recognized in the epigastric region. The prominence in the epigastric region or below the ribs in the mammary line is smooth and tense on palpation; sometimes fluctuation can be detected. In a moderate proportion of cases the so-called hydatid fremitus is elicited, if the left hand be placed over the tumor and another portion tapped quickly and forcibly with the right.

The tumor is painless, and there is no tenderness on pressure. The patient suffers from distention. There is interference with respiration, so that frequently he is compelled to sit up in bed in order to alleviate the dyspnoea. The general health is usually unaffected.

In some cases the cyst is in such relation to the hepatic duct as to cause compression jaundice. The jaundice usually develops gradually. In rare cases the cyst breaks into the hepatic duct; some pain follows this accident, and on account of the obstruction by the cystic contents jaundice develops. If the patient comes under observation after rupture of the cyst has taken place, the diagnosis is rendered more obscure. The enlarged cyst has been dispersed, and therefore most of the signs of tumor disappear.

Suppuration of the cyst sometimes takes place, and in addition to the symptoms due to hepatic pressure those of pyæmia arise,—rigors, periodical elevations of temperature, sweats, and great prostration. Jaundice occurs either because of the pyæmia, or, if it be intense, because of obstruction of the ducts and probably suppurative cholangitis.

The outcome of cases of hydatid disease varies. The liability to rupture is the same at all periods of life; perforation may take place into the stomach, the colon, the pleura and bronchi, or in some cases externally. It has been said that in a few cases where this accident has occurred recovery has taken place. The perforation may also take place into the pericardium or the vena cava; when this accident occurs death takes place suddenly.

Diagnosis.—A diagnosis is not usually difficult. Irregular enlargement of the liver, the surface of which is smooth and painless, or the presence of a tumor of the same character connected with the liver, probably fluctuating, in an individual otherwise in good health, usually indicates the presence of this disease. If the cysts are multiple, and the surface of the tumor, therefore, irregular, the diagnosis is more difficult. The health is usually retained, and the benign nature of the enlargement thus inferred. Syphilitic disease of the liver and carcinoma must be excluded in adults. The rarity of the latter affection in childhood and the absence of a primary focus of malignant disease, with retention of health and strength, exclude cancer. In syphilis the enlargement of the liver may be irregular and a distinct boss recognizable. This usually occurs in tertiary syphilis, a form not seen in childhood. In congenital syphilis involving the liver large prominences are not seen. Nevertheless, in both

instances it is well to resort to exploratory puncture, and, if syphilis be suspected, to the treatment as a test in diagnosis. If suppuration takes place in the cyst, it cannot be distinguished from abscess unless it be known before the accident that there was a painless enlargement of the liver without fever. In adults dilatation of the gall-bladder has been mistaken for hydatid. This condition does not occur in childhood, and hence need not be considered. Hydro-nephrosis has also been mistaken for hydatid disease. The condition is not common in children, but can be distinguished by the results of exploratory puncture. When the cyst extends upward, it is often difficult to distinguish it from a pleural effusion. The same physical signs in the lower part of the right chest may be present as in effusion. Frehrichs believed that the direction of the upper line of dulness is significant in hydatid disease of the liver. It does not take the S curve, as in effusions, but reaches the highest point at the angle of the scapula. Sometimes empyema complicates a hydatid cyst, as in cases reported by Murchison. The cases that are most difficult of diagnosis are those which have ruptured into the lungs before coming under observation. The appearance of hooklets in the sputum is characteristic.

Reference has been made in the beginning of this article to results of exploratory puncture in cases of suspected hydatid disease. The fluid withdrawn has special properties which render the recognition of the disease absolute.

Prognosis.—From results of observation at the post-mortem table we see that a number of cases of hydatid disease of the liver undergo spontaneous cure. These cases, of course, are not recognized during life. If the disease is recognized and the tumor is accessible, the prognosis is very good. The results of treatment are generally quite favorable.

Treatment.—Internal medication is of no avail and need not be discussed. Surgical procedures are necessary. Electrolysis has been used, but since the advent of antiseptic surgery has fallen into disuse. Medicated injections are not in high favor. Iodine, carbolic acid, solution of bichloride of mercury, and permanganate of potassium have been used, but the treatment is open to objections. Indeed, at the present time all methods except free incision are discarded as more or less dangerous. The uncertainty that attends the introduction of the trocar and the possibility of infection render such methods more or less hazardous, while the difficulty of completely emptying the cyst renders it liable to recur after the fluid is withdrawn. Recamier's method of opening into the cyst by caustics or the thermo-cautery has been employed. The method is tedious and painful, and not without danger.

The treatment by direct incision and evacuation of the contents of the cyst has been rendered possible by the developments of abdominal surgery. Incision should be made over the most prominent part of the tumor in the manner of performance of a laparotomy. After the cyst is exposed it should be attached to the edges of the abdominal incision; it is then opened by the knife and its contents evacuated. The daughter-cysts may be evacuated by forceps. Too much force must not be used. In order to secure complete evacuation irrigation of the cyst-cavity must be employed. A drainage-tube is then inserted and the patient dressed as in an abdominal operation. If the cyst grows from the upper surface of the liver, it must be evacuated by passing through the diaphragm. One or two ribs should be resected, the pleura stitched to the diaphragm, and evacuation then brought about by the previous method.

In cases that have been operated upon a form of urticaria known as the *hydatid rash* is sometimes seen. It is said that the fluid of a hydatid cyst

will not cause peritonitis. Any portions of the cyst-wall that are left behind or any of the daughter-cysts will cause suppuration.

CIRRHOSIS OF THE LIVER.

Through the writings of Palmer Howard, of Edwards, of Hatfield, and others we have learned that in its etiology, clinical course, and mode of termination cirrhosis of the liver in childhood does not differ from that in adult life.

Etiology.—Alcoholism is a very constant factor in its causation. The habit is usually fostered because of the delicate state of the child in early infancy, coupled with the belief of ignorant parents that rum contributes to its development. It is true some children from their swaddling-clothes have an appetite for liquor, and when not discouraged are likely to develop all the lesions of alcoholism. Syphilis, as already mentioned, is another prominent cause. In Howard's cases an adhesive pylephlebitis took place primarily, followed by secondary cirrhosis. In some of the recorded cases chronic heart disease was the causal factor. The infectious fevers, as scarlatina and measles, play an important part. Tuberculosis is attended by a form of cirrhosis both when the liver is involved in the tuberculous disease and independently of it. Howard and others believe that ptomaines and products of imperfect digestion may be productive of this affection. In rickets there is often found enlargement of the liver which is due to an overgrowth of connective tissue.

Hypertrophic or biliary cirrhosis is rarely seen in childhood. It is due to chronic obstruction of the biliary passages, and hence is present in congenital obliteration of the ducts.

From the recorded cases collected by the above-mentioned authors, cirrhosis of the liver has been found to occur more frequently in males than in females in the proportion of two to one. The largest number of cases occur between the ninth and thirteenth years. It is found, however, at birth, and may occur at any period subsequently.

Symptoms.—In the early stages of the disease capillary congestion is noted in the face. This may increase. As the disease advances the face becomes drawn, the parts free from stigmata are pale, or a sallow, muddy complexion is seen. The symptoms due to obstruction are usually most prominent. Gastro-intestinal catarrh is observed. Morning nausea and retching with discharge of mucus take place, the appetite is poor, the bowels irregular, alternating attacks of diarrhœa and constipation take place, and the bowel movements usually contain considerable mucus. Hæmorrhages from the lower end of the œsophagus, the stomach, or the intestinal tract may occur, and are very characteristic symptoms of cirrhosis. In gastric hæmorrhage the vomiting has no relation to food, and is not associated with symptoms of gastric ulcer. In the later stages of the disease hæmorrhages occur from the nose or the mouth, and purpuric spots develop. They are due to the state of the blood. Hæmorrhoids are frequently present.

Jaundice occurs in about the same degree of frequency as in the cases of adults. It is usually slight, and may disappear and recur two or three times in the course of the disease. Slight fever is seen in many cases. The temperature rises to 101° and 102° in the evening. It may be present for a long period of time, and as the end approaches disappear entirely.

The urine is high-colored, of high specific gravity, and contains an excess of urates and uric acid. Frequently nephritis develops in the course of the affection. Albumin is then found, and the urine contains hyaline and granular casts. The specific gravity always remains high, and there is an excess of

lithates. From time to time sugar may be detected, but a persistent glycosuria is not likely to arise.

On physical examination, when the disease is somewhat advanced, further evidences of portal obstruction and attempts at compensatory circulation are seen. The venules along the base of the thorax, extending across the chest in an arc, following the attachment of the diaphragm, are very distinct. The external veins, particularly the epigastric and mammary, are particularly distinct. If compensation does not take place, ascites develops, and after its development the feet may swell. The spleen is frequently enlarged, but its size often cannot be determined when ascites is present. The liver is found to be enlarged if the case is seen in the early stage, and it may be slightly tender on palpation. Subsequently it diminishes in size, or the small size is at once noted. The diminution of the left lobe is particularly noticeable. With the walls relaxed the edge and surface can sometimes be felt rough and granular. Some cases are not attended by atrophy. Thus there may be much fat in the liver, and, notwithstanding the connective-tissue overgrowth, the organ remains enlarged. Fatty atrophy of the liver is the name applied to this form. In "biliary cirrhosis" the liver is enlarged and smooth. Jaundice is permanent, and the other symptoms of cirrhosis are present.

On account of the organic disease of the liver auto-intoxication takes place with ptomaines or products of imperfect digestion. Low delirium, deepening into stupor, with the occurrence of frequent convulsions, or noisy delirium followed by convulsions, show the effect of the toxine on the nervous system. Jaundice is not necessarily present when these symptoms develop.

Diagnosis.—The disease may be far advanced, and not recognized because of the absence of symptoms or signs. A boy aged fifteen years died in the Presbyterian Hospital of typhoid fever. He had been under the observation of the writer for nine years. Acute rheumatic fever with endopericarditis was the reason of the first consultation; valvular disease continued. The patient had been in poor health, and the parents were wont to give him spirits. This had been continued more or less until the fatal illness occurred. At the autopsy cirrhosis of the liver in an advanced degree was discovered.

The appearance of the face, the symptoms of portal obstruction, and the physical signs of atrophied liver are points on which the diagnosis is based.

The occurrence of subacute gastritis with morning vomiting, of hæmatemesis, and of mælena, without the physical signs of a small liver, are nevertheless most suggestive, particularly if the patient be poorly nourished, with a drawn, pallid countenance, and especially congestion of the cheeks—venous stigmata. If ascites, enlargement of the spleen, and jaundice supervene, the diagnosis is absolute.

Treatment.—We can never tell whether the enlarged liver of the early stage of cirrhosis is one in which congestion predominates, or, on the other hand, one in which the overgrowth of connective tissue is in excess. If the former, we know that there are measures which markedly influence the engorgement. If the latter, it is possible a further increase may be averted by proper hygienic and prophylactic measures. It is our duty, notwithstanding the uncertainty, to relieve engorgement. External depletion by cups and leeches, purgatives in quantity to ensure three to six liquid stools a day, Rochelle salts, citrate of magnesium, and saline waters, are to be used. The waters of such springs as Saratoga and Bedford in this country, and Carlsbad in Germany, are beneficial. Counter-irritation in mild degree is likewise of value. If leeches or cups are inadvisable, stimulating liniments may be employed. The diet is to be carefully selected. A milk diet is for a time the most satisfactory.

Stimulants and rich, stimulating articles of food, fats, sugars, and starches are to be avoided. Waters are to be used abundantly; they may be taken hot in large bulk (a glassful) when the patient is fasting to flush the liver.

Phosphate of sodium may be advantageously added to waters to produce a depurative effect. At first small doses of calomel or mercury with chalk should be given for a few days. A furred tongue, nausea, constipation, with pasty stools, indicate its use. From time to time it should be repeated. Iodide of potassium has been said to relieve the engorged liver in the early stage of cirrhosis, but the chloride of ammonium is a better drug, in doses of five to ten grains in syrup of licorice or in emulsion, given every four hours.

The treatment of the second stage is entirely symptomatic. Gastro-intestinal catarrh, hæmorrhages, ascites, jaundice with its resulting phenomena, and finally the distressing symptoms of the cirrhotic cachexia, require in turn, or too frequently at the same time, careful therapeutic and dietetic management.

Whatever the symptoms may be, the diet plays a most important part. The class of food referred to above is to be selected; from time to time a strict course of milk diet may be instituted. Again, with the ascites most prominent, a dry diet should be advised. The condition of the stomach and bowels very largely determines the character of diet. If there be much intestinal dyspepsia, albuminoid food should be administered. Meats chopped fine and made into a pulp can be given for a long period of time. In order to create free discharge of the products of digestion, large quantities of water should be taken once or twice a day. The disadvantage of a continuous meat diet arises in the possible development of scurvy. This may be counteracted by the use of lemon-juice once or twice in twenty-four hours. The gastro-intestinal catarrh is treated by the same class of remedies as are indicated and have already been detailed in the management of catarrhal jaundice.

Hæmorrhage from the stomach is to be treated by rest, the use of cracked ice, the external application of the ice-bag, the administration of food by the rectum, and the use of astringents. An opiate should always be given to quiet the agitated patient. Morphine hypodermatically or dry on the tongue may be selected, or the deodorized tincture of opium combined with the chosen astringents used. Gallic acid is one of the preferable astringents; aromatic sulphuric acid may also be employed. Both should be given well diluted in iced water:

R_x. Tr. opii deodorat.

Acid. sulphuric. aromat. āā f̄3j.—M.

Sig. Eight to ten drops every two, three, or four hours, well diluted.

The acetate of lead alone or with bismuth is a valuable astringent. Hamamelis may be given in the form of the fluid extract well diluted; twenty drops is a sufficient dose, and may be given every one or two hours to a child of ten. Astringent preparations of iron usually are advised—the sulphate, the chloride, and the pernitrate. They should be given in small doses frequently repeated. If nausea and vomiting are not present, ergot might be used; the writer, however, has never had any benefit from its use; indeed, gallic acid and the aromatic sulphuric acid have been sufficient to control the bleeding. Intestinal hæmorrhage may be treated by astringents by the mouth or by enemata. If bleeding be from the rectum or the lower portion of the colon, weak solutions of alum or of salts of iron by enema are of special value. The solution should be cold if the bleeding is from hæmorrhoids. One-half drachm of Monsel's solution to three ounces of water are the proper propor-

tions for enemata of this character. Ice may be used in the rectum, as well as ice-water. By the mouth the astringents advised for gastric hæmorrhage can be used. It is best to administer them in such form that they will be dissolved in the intestine; a one-grain pill of Monsel's salt may be given every half hour or hour. The pill should be hard. Acetate of lead in pill may also be given. In this class of cases aromatic sulphuric acid has been sufficient in the writer's experience. Turpentine has been advised by competent authorities, and may be given in capsule in doses of two or three drops every two hours. The oil of erigeron is also considered to be a valuable styptic.

If the ascites be not too great or of too long standing, it may be removed by dry diet and diuretics. Alkaline diuretics are particularly of service. Cream-of-tartar lemonade and infusion of scoparius are excellent diuretics. Saline waters which act on the kidneys and the bowels are of great service. Gentle catharsis may be maintained without fear of exhaustion if salines be used. On account of the tendency to intestinal catarrh, irritating cathartics should not be employed. At times the effusion seems to come to a standstill; the bowels have been sluggish, and the internal viscera apparently loaded with stagnated blood from passive congestion. A brisk cathartic often relieves engorgement and starts up absorption of the exuded fluid. In children the compound jalap powder is the best of the class. It should be given in doses of twenty grains; the amount may be increased if necessary. If the simple diuretics and cathartics are of no avail, four measures are to be considered and may be tried:

1st. The use of calomel with diuretics, as in the well-known pill of calomel, digitalis, and squills. It may be given in accordance with the following formula:

Ry. Hydrarg. chlorid. mit.	gr. $\frac{1}{16}$.
Pulv. digitalis	gr. $\frac{1}{4}$.
Pulv. scillæ	gr. $\frac{1}{4}$ —M.

Ft. pil. No. i.

Sig. To be taken every three hours.

After this combination is administered for ten days it should be withdrawn and squills and digitalis given alone. It then may be resorted to again, the frequency of its use depending upon the effect of calomel on the bowels.

2d. Caffeine is a valuable diuretic, particularly if stimulating effects are desirable. Dose 1 to 3 grains to a child under ten. The hydrochlorate of cocaine is another drug of the same class, and seems to have been of service.

3d. Copaiba. This is a most valuable drug in ascites. Its diuretic effect is decisive and usually permanent; it is to be given in capsule; three minims is sufficient for a child, to be taken every four hours.

4th. Paracentesis. Paracentesis should be employed early and frequently, if after a short trial the remedies above indicated do not lessen the amount of effusion. No hesitancy should arise on account of danger, as no accidents or complications are likely to occur. A number of cases have been reported in which frequent tapping has cured the ascites, and thereby arrested for a time at least the progress of the hepatic disease.

The treatment of jaundice need not require further consideration, for it has been discussed fully in a previous portion of this article. The symptoms of the cirrhotic cachexia which ensue in the latter stages of this malady are alleviated by careful nursing, attention in detail to diet, and the administra-

tion of remedies which secure full functional activity of the various organs of the economy. This particularly applies to the circulation. Cardiac tonics are indicated. Stimulants should not be withheld, and now are of service to counteract prostration, aid digestion, and increase the strength of the heart and circulation. All measures that can be invoked to relieve exhaustion, improve anæmia, and aid nutrition should be resorted to. The administration of concentrated food—animal broths, meat extracts, etc.; the inhalation of oxygen; the use of stimulating baths and lotions; measures to prevent the development of bed-sores,—each or all may be used as indications demand. Proper clothing, in order that the extremities and abdomen may be kept warm, must be insisted upon. At this stage multiple hæmorrhages and purpura are liable to ensue. The internal administration of astringents, but more particularly of turpentine, or the oil of erigeron, appears to check their development.

PERITONITIS; TUMORS OF THE PERITONEUM AND OMENTUM; AND ASCITES.

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I. ACUTE PERITONITIS.

THIS affection is an acute inflammation of the serous membrane lining the abdominal cavity and covering the abdominal viscera. It is characterized by a tendency to effusion, by adhesions through coagulable lymph, and by the deposition of purulent or sero-purulent fluid. Such an inflammation may be confined to a portion of the membrane, when it is said to be circumscribed or local, or it may involve the whole surface of the peritoneum, and thus become general. At the onset only will it be circumscribed or limited, for, unless checked, it quickly manifests a disposition to extend over the whole of the inner surface of the peritoneal sac.

Etiology.—Peritonitis may occur during intra-uterine life, in the new-born, and during infancy and childhood. In early life idiopathic peritonitis is not a very frequent disease, since at this period the peritoneum is not so susceptible to inflammation as the serous membranes of the thoracic and cranial cavities. When it occurs during intra-uterine life, it is always traceable to syphilis in the parents. It may cause the death of the fœtus in utero, or the child may be born suffering from the disease or its consequences. So far as is known, no symptoms in the mother serve to indicate the existence of peritonitis in the fœtus. If it be not fatal before birth, the resulting adhesions are very apt to interfere with the development of the intestines or to cause a constriction of a portion of the bowel.

In the new-born, acute peritonitis is most frequently the result of septic or pyæmic processes. It is usually caused by an unhealthy inflammation of the umbilicus or by the absorption of septic matter at that point. (See Diseases of the New-born.)

In infancy and childhood an attack may be traced to exposure to wet and cold. Thus, wetting and chilling of the feet, damp beds, chilly winds, sudden alterations of temperature, rapid cooling of the heated body, and excessive fatigue may be enumerated under this head as causes of acute peritonitis, just as they may act in the production of inflammation in other structures. Very often traumatism may serve as the exciting cause, and here may be enumerated contusions, direct blows upon the abdomen, and the wounds of cutting or blunt instruments produced accidentally or surgically, as in paracentesis abdominis. Again, various mechanical causes (which are in reality traumatic in their nature) may operate in its production, such as intestinal invagination, strangulated hernia, displacements of some of the internal organs, or laceration or unusual stretching of the peritoneal membrane. In like manner, peritonitis

may be caused by the extrusion of foreign matters into the serous cavity, as in ruptured hepatic or splenic abscess, rupture or perforation of the stomach, bile-ducts, spleen, uterus, urinary bladder, ureters, or some part of the intestines. It may follow or accompany acute disease of some organ by contiguity of structure; and here may be mentioned gastritis, splenitis, hepatitis, dysentery, ulcerations in typhoid fever, and ulcerations of the vermiform appendix, appendicitis, and the like. Numerous instances have been encountered where an empyema perforating the diaphragm has set up acute peritonitis; and this result has been observed even in the absence of perforation, the lymphatics acting as the channel of communication. In girls purulent vulvo-vaginitis has frequently caused acute peritonitis by an extension of the inflammation through the uterus and Fallopian tubes. At times also it may result from pressure and ulcerative absorption caused by tumors and malignant growths. The sudden retrocession of a cutaneous eruption has sometimes been closely followed by an attack of peritonitis, and it is on record that lumbricoid worms have penetrated the bowel and thus acted as an exciting cause.

Finally, it may occur as a complication of, or a sequel to, rheumatism, erysipelas, pernicious intermittent fever, and the various exanthemata.

It has been quite well established that in the development of peritonitis micro-organisms, rendered operative by any of the before-mentioned local disturbances, must be regarded as the essential causes. When, experimentally, non-pathogenic microbes, even when combined with small amounts of chemical irritants, are injected into the peritoneal sac, purulent peritonitis is not produced, but only a serous inflammation. On the other hand, when pathogenic micro-organisms are introduced even in very small quantities, severe fibrinopurulent peritonitis ensues. The micro-organisms which produce peritonitis are those found in pus, the staphylococcus, and the streptococcus. Before they can increase in number a preceding or accompanying change in the peritonium is necessary. If the absorptive powers of the peritoneum be greatly changed, the microbes will effect a putrefaction of the intraperitoneal fluids, and as a consequence will produce a general putrid infection of the whole system.

Pathological Anatomy.—The transparent and shining appearance of the membrane is lost. This is accompanied by a diminution of the lubricating secretion, rendering the serous membrane less moist. The subperitoneal vessels become turgid with blood, are visible through the thin membrane as an interlacing network, and when they are greatly distended the peritoneum presents a velvety appearance. At times the blood exudes through the coats of the vessels, when puncta or plaques of sanguineous effusion are seen. Later, if the disease progresses, the serous secretion is increased in quantity and altered in character, being composed of liquid serum and a more solid or glutinous material known as coagulable lymph. It may happen, however, that the effusion is entirely composed of serum; or, on the other hand, serum may be altogether absent. In metastatic peritonitis or in attacks of asthenic character the effusion may be puriform or distinctly purulent, while in sthenic cases the deposit of lymph may vary from a very thin layer to a thickness of several lines, and it is usually of a yellowish color. When abundant, it may be found in layers, smooth or corrugated, or it may exist as bands of adhesion uniting the viscera with each other or with the parietal peritoneum. At first villous in appearance, it afterward becomes smooth and denser, and finally assumes a structure similar to true peritoneal membrane. When once formed, plastic lymph acts as an irritant to the serous surfaces with which it may come in contact—a fact which serves to explain how inflammation is so apt to be diffused over the entire

peritoneal surface. When health is restored these bands of adhesion may partly or entirely disappear. If they continue they may cause little inconvenience, though it may happen that, by their topographical relations, they may interfere with the functions of the organs to which they adhere. In children the effusion is most commonly purulent; it may be merely puriform, decidedly purulent, or simply sanious. Ulceration may occur through the abdominal walls or through the diaphragm into the lung or bronchi, or again through the digestive tract, the bladder, the vagina, or through the psoas muscle, permitting pus to escape from the peritoneal cavity by one of these various channels.

When peritonitis exists as a sequel to scarlet fever, measles, rheumatism, or other fever, the serous fluid is in excess, whilst the plastic lymph is considerable in amount or nearly absent. The results of an attack, while at times causative of further disease, may in other cases be protective against more serious accidents: adhesions may supervene which will seriously interfere with the functions of the organs or parts which are bound down or united by these bands; on the other hand, as in some cases of perforation, this same inclination to plastic exudation may be conservative of life, the deposit being a means by which nature seeks to effect reparation.

Symptoms.—The earliest and most pronounced symptom of peritonitis is pain. At first the area of pain may be limited; afterward it will extend over the whole abdomen. The pain is accompanied by high fever and decided constitutional disturbances, such as rigors and general malaise. Pressure over the abdomen and augmented action of the abdominal muscles, as in deep inspirations, coughing, sneezing, expectoration, and the like, will aggravate the pain. The lightest weight cannot be borne upon the abdomen; hence the little patient assumes a position which will relax the abdominal walls as much as possible, and lies quietly on his back with his knees bent and thighs flexed. The belly is hot, rounded, and tense, almost invariably swollen and tympanitic from accumulation of flatus due to paralysis of the muscular coat of the intestines. Sometimes flatus may be readily passed per anum, at others not; and in this case symptoms of intestinal obstruction are simulated. The bowels are usually constipated, though diarrhoea is occasionally met with. Vomiting is nearly always present from beginning to end, and is aggravated whenever food is taken, until the presence of bile and faecal matter in the ejecta may be almost suggestive of some mechanical bowel obstruction.

The skin is hot and dry; the temperature, as a rule, is elevated, ranging from 101° to 105° F., but it becomes subnormal if the attack terminates in collapse. Inflammation of the peritoneum, however, may coexist with a normal or subnormal temperature, and this very frequently happens in the purulent cases. The pulse is small, feeble, rapid, and wiry. The respirations are accelerated, short; incomplete, and jerky, and are costal in type, the abdominal wall remaining motionless. The tongue is coated and the breath is foul. The face is expressive of great suffering and anxiety, and when the attack is very severe, the alæ of the nose are drawn upward, the nostrils are dilated, and the lips are parted, so as to expose the teeth, producing the expression known as *risus sardonius*. The urine is scanty and high-colored, and often contains albumin.

When the attack is to terminate in recovery all these symptoms gradually diminish in intensity, whilst the countenance, which has been so truthful an exponent of the patient's condition, once more becomes placid and natural. If the attack is to eventuate in death, the pulse becomes quicker and more thready, the general surface cold and clammy, the extremities chilled, and the breathing

shallower and more rapid, until life goes out from failure of the general vital forces. In a few cases incoherency of speech or active delirium is present toward the end, but most generally the mind remains clear and logical to the last. The fatal issue of an attack may take place in two or three days, though frequently the patient may live until the sixth, seventh, or eighth day.

Diagnosis.—Peritonitis in its severer forms is readily recognized, but when subacute or circumscribed, or when it is secondary or exists as a complication, it is not so easy of diagnosis. In young children it is especially difficult to determine its presence because of the uncertainty of exact localization of pain. In erratic cases also pain may be absent, and thus we will be hampered in diagnosis. The diseases simulating acute peritonitis are gastritis, enteritis, colic, rheumatism, neuralgia, renal calculus, and lead-poisoning. The diagnosis must depend upon the severity of the symptoms, special attention having been paid to the history of the case. If there be persistent vomiting of all fluids and solids, with the presence of sharp paroxysmal pain, accompanied by tenderness on pressure upon the abdomen, with cessation of the abdominal respiratory movements, a frequent, wiry pulse, and fever, the diagnosis of acute peritonitis may be made with reasonable certainty.

Prognosis.—In the generalized form prognosis is always grave. It has been said that there is no more fatal disease, recovery taking place in rare cases only. The more pronounced the symptoms are, the more doubtful will be the prospect of recovery; and if the patient shall have passed into the stage of collapse, a fatal termination is usually to be expected. An acute peritonitis which is metastatic in origin or which is due to perforation is generally fatal. Diarrhœa is of evil portent, and constant vomiting with complete obstruction of the bowels is a very grave symptom.

Although the general prospects of recovery are so slight, yet patients apparently moribund have been known to get well. The attack may last but a few days, or even only from thirty-six to forty-eight hours, and very rarely indeed longer than a week.

Treatment.—The cause of the attack will determine the treatment to be followed in a given case. Unfortunately, however, the physician will not always be in a position to know accurately what this may be or what exact anatomical lesions may exist. The patient must be confined absolutely to bed. All food and drink must be stopped, only cracked ice or iced water to moisten the mouth being permitted. This interdiction of all ingesta must be imperative, in order to avoid the harassing and painful vomiting. Nutrition can be maintained until the cessation of vomiting by the use of enemata or suppositories containing appropriate substances, as broth, milk, egg-albumin, stimulants; later, when the vomiting shall have been overcome, the food should be limited to twelve or fifteen ounces (best predigested) per diem. Ice pills may be given to control the vomiting, also iced champagne in small doses frequently repeated, as well to soothe the feeble stomach as for its stimulating effects. Locally, various remedies have been employed. Soft flannel cloths saturated in a solution of tincture of iodine in castor oil and applied over the belly have been highly recommended. Local bloodletting by the application of from four to twelve leeches to the surface of the abdomen is often very valuable in the initial stage. The inunction of mercurial ointment to the abdomen was formerly much in vogue. Stupes, made by steeping flannels in a pint of hot water containing ten to twenty drops of spirits of turpentine and sprinkled with laudanum, are often of great service. Light flaxseed-meal poultices, dashed with oil of turpentine or laudanum and laid upon the abdomen, have in my hands been of great value. Care should be taken that the poultices be

not too hot, lest the integument be burned. By some physicians cold applications, such as the ice-bag or cold-water coil, are preferred, but children almost always resist their use.

As to the methods of internal treatment—whether by saline purgatives or by opium—a difference of opinion still exists among physicians of equal skill and eminence. It will be safe to abide by the following conclusion: When an attack of acute peritonitis is recognized almost at the moment of its inception, salines by their rapid and complete depletion may abort an attack. The peritoneum will be drained of the products of inflammation, the formation of bands and adhesions will be prevented in consequence of the increased peristaltic action of the bowels, whilst, clinically, pain will be relieved as quickly as by the administration of opium. On the other hand, if the case is not seen by the physician until some hours after the commencement of the attack, and especially if grave doubts exist as to the cause of the disease, opium and external methods of depletion must be used. It need scarcely be said that in perforative peritonitis the purgative treatment must not be thought of at all.

In case it has been decided to administer a purgative, either a seidlitz powder or some other mild saline or calomel is to be preferred for children. When opium is to be given—which should always be the case when there is intense pain, tenderness, constant vomiting, and a distended and paralyzed condition of the bowels—it should be given in quantity sufficient to relieve pain, to reduce the frequency of the pulse and respiration (the latter to about twelve movements per minute), and to make the little patient slightly drowsy. Two to five minims of the deodorized tincture of opium, or one to four grains of Dover's powder, may be given every four hours, according to indications, at the age of six years. The effects of the opium must, of course, be narrowly watched, for, as is well known, children are very susceptible to its action. In older children morphine may be given either *per ore* or by hypodermatic injection in doses of from one-twelfth to one-sixth of a grain. The tincture of belladonna is frequently combined with the opium. Excessive tympany can be relieved by the use of laxative enemata in which spirits of turpentine or tincture of asafoetida has been suspended; or, in case of their failure, the long rectal tube may be used. Free stimulation must be resorted to early, and such alcoholics as brandy, whiskey, and champagne are to be preferred. To these may be added, to assist in keeping active a flagging circulation, such cardiac stimulants as sparteine, strophanthus, and digitalis; these, if vomited, must be given by the rectum or under the skin. Later, when the attack promises to terminate favorably, every effort must be made to build up the system and to increase nutrition by the exhibition of tonics and easily-assimilated, nourishing food.

As soon as the diagnosis of acute peritonitis has been made, the question of opening and draining the peritoneal cavity will present itself. Here, again, differences of opinion are encountered. Some advocate an early and immediate operation, whilst others claim that, as cases recover without operation, it is better not to risk the added dangers of surgical interference. It may be considered proper to operate in the following forms of peritonitis: First, in the fulminating forms of the disease, which are characterized by a rapid advance of the symptoms, excessive vomiting and tympanites, feeble pulse, and great restlessness. Secondly, in cases in which collapse seems imminent in spite of treatment, and which present a decreasing temperature and a rapid pulse constantly growing feebler. Thirdly, in cases in which pus is present in the abdominal cavity, or in which a tumor is located in, or adjacent to, the abdomen. Fourthly, in cases in which the peritonitis is the result of perforation or ulceration of any of the abdominal viscera. And fifthly, when the peritonitis is

due to intestinal obstruction. In older children the chances of success are greater than in the younger ones. A certain number of cases will be met with in which the diagnosis will be questionable. It will, at times, be doubtful whether the exudation be purulent or composed only of lymph, and whether the inflammation has been general from the first, or has spread from the cæcum or other localized inflammatory area. In such cases it is held to be justifiable to perform an exploratory operation, which may, under some circumstances, be the means of saving the patient's life. To discuss the method of operation and the questions of drainage and irrigation is not embraced in the scope of this article, and for such details the reader is referred to the works on operative surgery.

II. CHRONIC PERITONITIS.

As a chronic affection peritonitis, with the exception of the tubercular variety, is rare. As early as 1838, Wolff published a study upon chronic peritonitis, and stated it to be an extremely common affection in children; but as all of his one hundred cases were reported cured, it seems likely that a large proportion were incorrectly diagnosticated. Since then until quite lately the subject has received but little consideration at the hands of medical writers, and the opinion has gained ground that all chronic peritonitis, almost without exception, is tubercular (West). This view, however, has been considerably modified by the more recent studies of Baginsky, Vierordt, Henoch, and others, and it is now accepted that the peritoneum, just as well as the pleura, may be the seat of a simple chronic inflammation with serous exudation.

Chronic peritonitis may sometimes be the sequel of an acute attack of the disease, but it is more frequently an independent affection.

Etiology.—Most of the patients are females—a fact that suggests a possible connection in some cases between the peritoneal inflammation and a vulvovaginitis, which is by no means uncommon in little girls. Rarely a history of traumatism may be elicited, as in a case reported by Henoch, confirmed by post-mortem after a course of six weeks. In another group of cases a preceding exanthem may be the apparent etiological factor, as seemed to be likely in two cases—one observed by Fiedler, and the other by Henoch—both occurring after measles. The complete cure, after several tappings, in Henoch's case leaves little doubt of its true character.

Symptoms.—The symptoms of non-tubercular chronic peritonitis are rather obscure. The abdominal pain is apt to be slight, whilst the constitutional symptoms are variable. Usually the health fails gradually; the appetite becomes capricious; there is alternate diarrhœa and constipation, the former of which may or may not be accompanied by pain; sleep is disturbed, and the skin is hot and dry at night. Subsequently, pain or a sensation of tightness in the abdomen is complained of, and after a time effusion of fluid takes place, fluctuation may be discovered on examination, and the cutaneous veins are turgid and well defined. The pain now becomes more marked; it is usually not localized, but shifts about from one spot to another; generally there is tenderness on pressure over the abdomen; still, the appetite may be fairly good, the tongue tolerably clean, and the bowels not particularly irregular. As the effusion accumulates dyspnœa appears; the pulse is accelerated; evening and morning exacerbations of temperature are observed; the child rapidly loses strength, becomes much emaciated from profuse diarrhœa, and eventually dies of exhaustion. Yet cases presenting all the symptoms of chronic peritonitis have been known to recover, the effused fluid and other products of inflammation being gradually removed by absorption.

Diagnosis.—When ascites is the only symptom, it will be necessary to differentiate between an effusion due to simple chronic peritonitis and one caused by obstruction to the portal circulation. The latter condition is comparatively rare in childhood, whether it be due to cirrhosis of the liver or adherent pericardium and mediastinitis; and the chances are immensely in favor of the presence of a chronic peritonitis. The ascites due to cardiac disease can be eliminated by careful examination of the heart. Since, in the beginning of the disease, the symptoms simulate those of chronic intestinal catarrh, one must be careful to distinguish between this affection and chronic peritonitis.

The differential diagnosis between chronic and tubercular peritonitis will very often be impossible. The point of greatest value, however, is the general state of the patient: in the simple form the general nutrition and well-being of the child suffer but little as long as digestion is not greatly disturbed nor the effusion overwhelming; while in the tubercular variety the early emaciation is striking. Search for bacilli in the effusion, even in tubercular cases, is often disappointing, and hence a negative finding does not exclude the more serious disease.

Prognosis.—This must be guarded, for, while most cases are decidedly unpromising, a certain proportion recover. The history and progress of a given case must give us the cue.

Treatment.—As the disease usually begins with an intestinal catarrh, our treatment must be directed toward that condition. The child must be placed under the best hygienic surroundings. Plenty of sunlight and, when possible, country air or a sojourn at the seashore, are to be insisted upon. The clothing should be carefully regulated to meet the exigencies of the case, the weather, and other external conditions; and a flannel bandage must be constantly worn about the abdomen. The patient should be kept at rest, and it is a good plan, in the warm weather, to wheel his couch into the open air as often as possible.

The diet should be bland, but nutritious. Moderate quantities of underdone chops or steak, fish, fowl, and eggs are all allowable; so also are milk and cream if they do not disagree, but starchy foods are better avoided.

Abdominal pain may be relieved by hot opium fomentations or inunctions of belladonna ointment; when these fail or in protracted cases, blisters or stimulating liniments, tincture of iodine, compound iodine ointment, and iodide of potassium ointment are useful applications. Frequently in these cases the application to the abdomen of a mild mercurial preparation, such as an ointment of the yellow oxide of mercury, about twenty grains to the ounce, will do good service.

In the way of medicines the mineral acids and preparations of pepsin are useful as aids to gastric digestion; and to combat the intestinal catarrh, bismuth, sulpho-carbolate of zinc, the bitter vegetable tonics, and alkalies should be administered.

The internal use of iodine is also beneficial. This may be administered in the form of iodide of potassium in guarded doses, which must be discontinued on the first indication of disordered digestion; but a preferable form is the syrup of the iodide of iron, in doses of from five to thirty drops, according to the age and tolerance of the patient, several times daily. I usually order it to be given in cod-liver oil, which is convenient and efficacious.

If the ascitic effusion shows no tendency to disappear by absorption, tapping by means of a very small trocar and canula should be resorted to, the fluid being allowed to drain away very slowly. It has been advised that during the first few weeks the fluid be drawn off once in twenty-four hours, the amount varying in quantity from one to two pints; then every two, every three days,

and, finally, once a week. Gradual improvement, it is said, usually takes place under this treatment. When the disease has defied every method of treatment, especially if the fluid returns quickly after repeated tapplings, permanent drainage of the peritoneal cavity has been recommended. If pus be present, incision and drainage should be practised. Recently cœliotomy and washing out of the peritoneal cavity have been advocated by some surgeons as a routine treatment. In some instances it may even be justifiable to perform an exploratory operation. However, in those chronic cases in which the symptoms are not urgent and the child is not failing, it will be the part of wisdom and prudence not to interfere surgically, but to wait on nature's efforts, supplemented by medical measures, to effect a restoration to health.

III. TUMORS OF THE PERITONEUM AND OMENTUM.

Tumors of the peritoneum and omentum, though rare in children, are occasionally met with.

Carcinoma of the peritoneum has been encountered in early childhood and even in foetal life. It may be primary, and then is often congenital, but it is much more commonly secondary. Scirrhus is the usual variety, and generally occurs in diffused nodules. The primary form is difficult to detect; the secondary, much less so, because its presence will be suspected when symptoms referable to the peritoneal cavity occur in the course of cancerous invasion of some other portion of the body.

Sarcoma of the peritoneum has also been met with in childhood, but it is of very rare occurrence. This variety of neoplasm may grow to such an extent as to involve the omentum, mesentery, and other parts in addition to the peritoneum; in fact, both carcinomatous and sarcomatous growths are apt to involve both peritoneum and omentum.

Lipomata may also grow from the peritoneum. They are encapsulated, and have no connection with any other organ.

Serous cystic tumors of the peritoneum also occur. These cysts are composed of pseudo-membrane, which during their evolution and organization includes a portion of the fluid exudation and receives an internal serous investment; they are attached to the peritoneum either by means of a thin neck or by a broad base. Cystic tumors of the peritoneum are difficult to detect, and must be differentiated from cysts of the omentum, from cysts of the various abdominal viscera, and from ascites.

Tumors of the omentum are quite rare in early life. Omental cancer is usually of the colloid variety, and it may grow to an enormous size. Carcinoma, however, is seldom limited to the omentum, the peritoneum being usually involved simultaneously. Again, when scirrhus invades the peritoneum the omentum usually suffers from the same disease. Cysts and hydatid tumors of the omentum are met with in children, the former not infrequently. The cysts are usually dermoid in nature, though simple serous cysts are encountered. Both varieties, but especially the dermoid, may suppurate.

Symptoms of all varieties of tumors, whether involving the omentum or the peritoneum, or both, are rather vague, particularly in their incipency. Later, when they have grown larger, the so-called pressure-symptoms develop and aid us in making a diagnosis. Even then it is very difficult to make a correct differential diagnosis, the pressure-symptoms chiefly aiding in locating the site of the tumor, without throwing light upon its character.

Cancer of the peritoneum and omentum produces the signs of a diffuse, more or less acute peritonitis with effusion, the so-called cancerous peritonitis.

In the earlier stages of the disease the patient will complain of paroxysmal pain, which later will be more persistent. Lipomata are attended by no special symptoms beyond the growth of a painless tumor. Growths confined to the omentum are movable and occasion no functional disturbance of the intestines. In cystic tumors, either of the omentum or peritoneum, the abdomen is apt to be enlarged; if the tumor be superficial, it will be movable on palpation and give signs of fluctuation, which must be distinguished from the fluctuation of ascites. If the patient live long enough and the growth attain the proper size, true ascites will supervene. Pain, of course, will be most prominent in the cases of cancerous tumors. In time, whatever may be the nature of the tumor, but particularly in the cases of carcinoma, the general system suffers, nutrition is impaired, the patient is easily fatigued, his appetite fails, and, if the growth cannot be removed, a cachectic condition develops which at last terminates in death.

Prognosis is most favorable in cystic tumors, less so in lipomatous and hydatid growths, and fatal in the carcinomatous.

Treatment of cancerous tumors consists mainly of palliation of symptoms. Anodynes and opiates to control the pain are indicated, and, if the ascites become burdensome, paracentesis is to be performed. Attention to the general condition of the patient, sustaining his strength with good food and tonics, together with the observance of well-established hygienic principles, will embrace all that can be done for these unfortunate sufferers. Operative measures are not to be advised in these cases.

Operation, cœliotomy, has been more successful in cases of sarcoma, lipoma, hydatid growths, and particularly in cystic tumors. Cystic tumors may be excised or they may be aspirated and drained. As drained cysts are apt to refill, the radical operation, excision, is to be preferred, and it must always be resorted to when supuration takes place.

The proper treatment for pressure-symptoms will be suggested by their characters in individual cases.

IV. ASCITES.

Ascites is an accumulation of fluid—usually serous—within the peritoneal cavity; occasionally chylous ascites occurs, but in children this variety is extremely rare. Essentially considered, ascites is not a disease. It is a symptom of either general dropsy or some local disease of the abdominal viscera, and consists of a transudation of liquid into the peritoneal cavity in consequence of disturbed circulation in the liver or of pressure exerted upon some portion of the portal circulatory system.

Etiology.—The most common cause of ascites in children is cirrhosis of the liver, which, in turn, is most frequently due to syphilis. It may also arise from a simple osmosis of the watery constituents of the blood, in which case it is but a local expression of a general hydræmia superinduced by some cachexia, and it is then often associated with hydrothorax or general anasarca. Again, it may result mechanically from an obstruction to the venous circulation caused by cardiac, pulmonary, or peritoneal disease. Neoplasms of the abdominal cavity, whether malignant or benign, and particularly lymphatic tumors situated in the hilum of the liver, will also cause it by mechanical interference with the circulation in the viscera.

Bright's disease and acute nephritis; organic heart disease; atelectasis pulmonum and emphysema; enlargement of the spleen and profound anæmia caused by malarial poisoning; the pressure of lardaceous lymph-glands upon

the portal vein and inferior cava; and occasionally chronic tubercular peritonitis, which interferes with the circulation in the peritoneum,—are other etiological factors. Interstitial nephritis is not so apt to cause an ascites as a general anasarca. Benign tumors in the abdominal cavity are exceptionally accompanied by ascites, malignant tumors constantly.

Pathology.—The pathology of ascites is comprehended in the lesions involved in the primary affection. The changes in the peritoneum itself are slight and inconstant. Sometimes this membrane has simply a reddened appearance, but not infrequently it is pale and devoid of any signs of inflammation.

Symptoms.—The constitutional disturbance attending the formation of an abdominal effusion usually passes unnoticed, but it may be ushered in with chilliness, nausea, headache, vomiting, colicky pain, or a brief, intermitting diarrhoea. Pain is absent unless the effusion is caused by peritonitis. When effusion has reached a certain point, the tenseness of the abdominal walls is apt to cause indigestion and irregularity of the bowels; the skin becomes dry and has an ashen or clayey look; and the navel protrudes and may be encircled by a plexus of dilated veins, termed “caput Medusæ.” In very large effusions the skin of the abdomen becomes stretched and glistening, and at times fine white striæ, similar to those which are observed upon the abdomen of a pregnant woman, make their appearance. The character of the pulse depends upon the primary disease; still, it is generally feeble and easily compressed. Provided no inflammatory disease coexists, the temperature is normal. The urine is variable in quantity, though usually diminished; then it is high in color, and may contain albumin and fibrinous casts. As a result of mechanical interference with the return circulation from the lower extremities ascites is very frequently attended by œdema of the feet and ankles. Large effusions, crowding against the liver, spleen, and kidneys, and forcing the diaphragm up to the second and third ribs, cause anæmia of these organs and collapse of the base of the lungs, with consequent general anasarca.

Finally, painful and difficult micturition or incontinence of urine, together with difficulty in evacuating the bowels, will ensue. The constant crowding upward of the diaphragm and liver causes dyspnœa, hydrothorax supervenes, and at last the child, unable longer to assume a horizontal position, dies either from asthenia or asphyxia.

Physical Examination.—Palpation and percussion reveal fluctuation indicative of the presence of fluid, which varies in position according to the posture assumed by the patient. Thus, whilst standing, the abdomen is largest in its lowest part; when prone it spreads laterally, and if the patient be turned on either side it falls toward the more dependent. In any of these positions percussion practised over the uppermost part of the abdomen, to which the gas-containing intestines always float if entirely free to move, gives a clear tympanitic note, and by successively altering the patient's posture the tympany readily moves from point to point, while the dulness due to the fluid also changes its place. Wave-like fluctuation is another valuable sign.

Diagnosis.—The diagnosis of ascites is comparatively easy, yet it must not be forgotten that in children other conditions are often encountered which produce an enlargement of the abdomen. Naturally, the smaller the effusion the more difficult is it to make a diagnosis. When the abdomen is distended by a sufficiently large amount of fluid, wave-like fluctuation and movable dulness can readily be obtained, and leave no doubt of the diagnosis. Small effusions, although always obscure, are most readily detected when the patient sits or lies on one side.

In addition to detecting the presence of ascites, it is necessary to determine

the nature of the antecedent disease, as upon this prognosis depends. When the fluid is large in amount and movable, atrophic cirrhosis of the liver may be suspected. If the effusion be small and immovable and loculated, the cause is most probably tubercular peritonitis. This disease is characterized by the presence of disseminated nodules, and its symptoms are tenderness upon pressure, pain, and fever, possibly conjoined with indications of tubercular disease in some other organ.

Prognosis.—Though not always a hopeless condition, the prognosis is not very encouraging. Provided the primary cause upon which the ascites depends be removable, as in malaria or alcoholism, and the liver is not completely invaded by the disease, we may hope, by removal of that cause and by proper treatment and hygiene, to effect a cure of the abdominal dropsy.

Treatment.—In the milder degrees of ascites treatment consists in the administration of diuretics, diaphoretics, and hydragogue cathartics. Acetate of potassium, combined with digitalis and compound spirit of juniper, acts favorably. A very efficient combination is the following :

R̄. Magnesii sulphat.
Potassii bitartrat. āā ʒss.
Aquæ cinnamomi fʒiii.—M.

Sig. A tablespoonful every three or four hours, according to its effect upon the bowels.

When great general anasarca coexists with the ascites, threatening interference with respiration and circulation, in addition to the free purgation hot vapor baths are to be recommended. These may be applied in the following manner: The patient, completely divested of clothing, is laid upon a blanket, and immediately several bricks, which have been in the mean time thoroughly heated by immersion in pails of hot water, and then enveloped in flannel cloths, are placed at the shoulders and feet. Care must be taken that they be neither too hot nor put too near the body, lest the skin be scorched. Another blanket is then thrown over the patient. The upper corners of the superimposed blanket are brought over and tucked under the opposite shoulders, while the other end of the upper blanket, with the lower end of the underlying one, are lapped together under the heels of the patient, and the head alone is left to protrude from this improvised sack. This hot pack is maintained for at least twenty minutes, producing profuse diaphoresis and usually greatly ameliorating the symptoms. The patient and his friends are apt to complain loudly of this heroic treatment, but I can recollect several instances where by its use the child was saved from imminent death; and often it will accomplish the end sought when all other measures have failed.

A strict milk diet is to be enjoined as a rule. When, however, hydræmia is prominent, iron, tonics, nutritious food, and good air, with a proper observance of all recognized hygienic rules, are indicated. In ascites depending upon atrophic hepatic cirrhosis squills, digitalis, calomel, and iodide of potassium will be of service. In this variety, however, the ordinary diuretics usually have but little effect. Here Basham's iron mixture is highly spoken of—viz.:

R̄. Tinct. ferri chlorid.
Acid. acetic. dil. āā fʒj.
Liq. ammonii acetat. fʒvi.
Aquæ q. s. ad fʒvj.—M.

Sig. Tablespoonful three times daily for a child of six years.

If, despite this treatment, the fluid continues to accumulate, paracentesis abdominis must be practised. This operation should not be performed too soon, nor should we delay it to the last moment. The proper time is when remedies fail after a fair trial and when, in spite of treatment, the patient's general health daily deteriorates. Ordinarily this operation is simple and free from danger. Either an aspirator or fine trocar and canula may be used, but I prefer the latter. This tapping can be repeated as often as the exigencies of each particular case may require. If fluid reaccumulates within three or four days, a retapping should be postponed as long as possible; if, however, a number of weeks elapse before the peritoneal cavity is refilled, the operation may be correspondingly deferred to that time.

Permanent drainage by means of a rubber tube under proper antiseptic precautions has been highly commended by Dr. A. Caillé, whenever, after one or two tapplings, the ascitic fluid rapidly reaccumulates. When all other measures of treatment are futile, this method of permanent drainage should be utilized.

While the operation of paracentesis is very trifling, every antiseptic precaution should be employed. In order to produce local anæsthesia a hypodermatic injection of three to five minims of a 2 to 4 per cent. solution of cocaine may be made at the proposed point of operation, or the same result may be obtained by the rhigolene spray or the application of ice and salt. The linea alba, below the umbilicus, is the usual point of election except for loculated effusions. In the latter case, as distended veins ramify extensively over the abdominal wall, caution must be used not to wound any of them with the trocar. As the fluid escapes pressure is kept up by means of a many-tailed bandage: this lessens the risk of syncope and secures a thorough evacuation of the fluid. If the puncture has been made at the side of the abdomen, the patient must lie on the opposite side for some little time, so that the wound may cicatrize properly. This will obviate the occurrence of a fistula, a sequel which will prove a source of great annoyance to the patient, inasmuch as leakage soils the clothing and provokes cutaneous inflammation.

CONGENITAL INTESTINAL MALFORMATIONS

AND

DISEASES OF THE ANUS AND RECTUM.

BY HENRY R. WHARTON, M. D.,
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I. CONGENITAL MALFORMATIONS OF THE INTESTINES.

CONGENITAL MALFORMATIONS of the small intestine are met with much less frequently than those of the rectum and anus; in the Vienna Foundling Hospital only 9 anomalies of this nature were found among 150,000 infants. The malformation may consist of a stenosis or atresia of the gut; or the bowel may terminate in a cul-de-sac at the point of obstruction, and beyond this point again begin in a cul-de-sac, the remaining portion of the intestine being well developed; or the bowel may have a diverticulum given off which attaches it to the abdominal walls, and this may contain a fistula opening upon some portion of the body; or, finally, the defect may consist in an abnormal shortness of the intestinal canal. Holmes mentions two cases of congenital occlusion of the small intestine in which the diagnosis was satisfactorily established, and Dr. W. Craig reports a case of congenital malformation of the small intestine in a child who lived seventy-two hours, and in whom the autopsy showed an obstruction of the small intestine at the upper fifth of the ileum. The intestine in this case was distended above the point of obstruction, and upon opening the bowel it was found that it ended in a cul-de-sac; further examination of the gut beyond the point of obstruction showed that the intestine began in a cul-de-sac, and the intervening space between these two pouches was occupied by a band of fibrous tissue. The most frequent position of congenital occlusion of the small intestine is the duodenum near the point at which the biliary duct and pancreatic duct open, or at the point where the duodenum becomes jejunum under the transverse mesocolon. Malformations of the ileum are most common near the ileo-cæcal valve, or a short distance above it, where the ductus omphalo-mesentericus is given off. Among the congenital malformations of the small intestine may be mentioned that condition known as Meckel's diverticulum, which consists in a cylindrical or flask-shaped appendage attached to the ileum a metre or more above the ileo-cæcal valve, and is a remnant of the omphalo-mesenteric duct. Another form of this defect consists in the presence at the umbilicus of a reddish tumor covered with mucous membrane, which has been described as a *warty tumor of the umbilicus*, *congenital mucous polypus of the umbilicus*, and as *adenoma of the umbilicus*.

Congenital malformations of the large intestine are also of infrequent occurrence, but may involve the colon, the sigmoid flexure, or the rectum. The malformations of the large intestine may consist of an occlusion of the

gut at any portion of its length; or the gut may exist in a rudimentary condition. The latter defect is most apt to be associated with Meckel's diverticulum, with a faecal fistula between the ileum and the fissure above the umbilicus, or with a faecal fistula between the small intestine and some portion of the abdominal walls. Atkin reports the case of a child who died two days after birth, and in whom, upon autopsy, the rectum and colon were found to be in a rudimentary state, smaller than an ordinary quill; in this case the parts had remained in the condition in which they exist in the early embryo.

The various congenital malformations of the small and large intestine are probably largely to be attributed to accidents in development due to a complicated disposition of the intestinal tract of the embryo; and it is also likely that foetal peritonitis plays an important part in the production of these deformities. There is of the opinion that many of these anomalies are due to changes in the peritoneum which have taken place early in foetal life.

Symptoms.—The symptoms arising from congenital malformations of the large or small intestine are simply those of intestinal obstruction in a more or less marked degree, which depends upon the completeness of the occlusion; and all observers are agreed as to the absence of any definite symptoms accurately localizing the seat of the lesion. The vomiting of whitish mucus, with obstruction of the bowels, in the case of a new-born infant, points to an occlusion high up in the small intestine, and if the obstruction exists in the jejunum or ileum, this may be replaced by the vomiting of meconium. In such a case the symptoms would in no wise differ from those consequent upon the presence of an occlusion situated in the region of the rectum or anus. If a faecal fistula is present, the symptoms of obstruction will not be so marked, and the position of the fistula may serve as a guide to the situation of an intestinal malformation.

Diagnosis.—As before stated, the localization of the lesion is often most difficult. In a newly-born child who presents swelling of the belly with vomiting and obstruction of the bowels, the anus and rectum should first be examined to exclude the possibility of malformation of these parts; a soft catheter should be passed into the rectum, and if, upon injecting water, meconium is brought away, it can be inferred that the obstruction exists at a higher point of the intestinal canal.

Prognosis.—The prognosis is always unfavorable: complete occlusions of the duodenum or of the high portion of the jejunum must necessarily prove fatal in a short time; but when the obstruction is incomplete or occupies a position low down in the small intestine, or if associated with a faecal fistula, the patient may survive for some time, even for years. Complete occlusions are usually fatal within a few days unless relieved by operative treatment.

Treatment.—In cases of complete obstruction operative treatment must be resorted to promptly. Up to the present time the results obtained have not been encouraging; but with the improved technique of abdominal operations more favorable results may be looked for in these cases. As before stated, the diagnosis of the seat of the lesion is often impossible; but as in cases of complete occlusion the result is necessarily speedily fatal, it seems wise to attempt an exploratory operation with the hope of affording relief or bringing about a cure. A median laparotomy, unless there is some definite symptom present which points to the exact seat of the obstruction, should be the operation selected. If upon opening the abdomen the occlusion is found situated in the duodenum or high up in the jejunum, the case must be abandoned as hopeless, unless it be found possible to excise the occluded portion of the bowel and bring the ends together by sutures (circular enterorrhaphy), or to make

an attempt to establish the continuity by the procedure known as lateral intestinal anastomosis. If the occlusion is due to a membranous septum, this may be exposed by incising the gut, and after it has been perforated or cut away the intestinal wound should be united by Lembert's sutures and the abdominal incision closed in the usual manner. If the occlusion exists low down in the small intestine or in the large intestine, circular enterorrhaphy or lateral anastomosis may be employed, or an artificial anus may be made by bringing the gut to the abdominal wound, securing it there, and opening it. This latter procedure would seem to be the wiser one, as it requires much less time to accomplish it, and if the patient survives, after he has attained some age an attempt may be made to establish the continuity of the intestinal canal by lateral anastomosis. If a fecal fistula is present and there are no marked symptoms of intestinal obstruction, no operative treatment should be instituted; but if the patient exhibits symptoms of intestinal obstruction, the fistula should be dilated or incised, and, if relief be obtained, further operative treatment should be postponed until a later period.

II. CONGENITAL MALFORMATIONS OF THE RECTUM AND ANUS.

Congenital malformations of the rectum or anus occur, according to various observers, in the proportion of 1 case in 10,000 births.

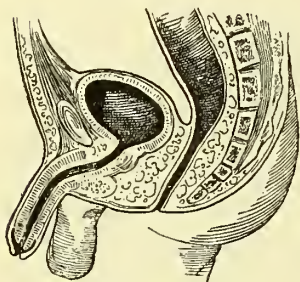
Pathology.—These malformations result from arrested development of the parts in early foetal life. At its earliest commencement the alimentary canal consists of a simple sac or bag developed from the innermost layer of the blastoderm, partly within and partly without the body; and as development proceeds this communication between the two portions of the sac is shut off, and the portion within the abdomen consists of a simple tube, the *mesenteron*, which terminates at the anterior extremity of the embryo in a blind pouch, while at the posterior extremity a similar pouch is formed. The cul-de-sac at the anterior extremity of the embryo comes in contact and communicates with an invagination of the epiblast, which is called the *stomodæum*, while a similar depression of the epiblast at the posterior extremity of the embryo, named the *proctodæum*, forms the anal orifice and communicates with the mesenteron. The majority of malformations of the rectum and anus are due to an interruption in the latter stages of the process just described, or, in other words, to an arrested or irregular development of the proctodæum or mesenteron. The termination of the rectum in the genito-urinary tract is due, in addition to the arrested development just mentioned, to a similar arrested development in the perineal septum, which separates the rectum from the genito-urinary tract, both, in the early life of the embryo, having a common orifice. The failure of development of the perineal septum explains the frequency of cases of imperforate rectum and anus in which there is a communication between the intestinal tube and the genito-urinary tract.

The best classification of the malformations of the rectum and anus is that adopted by Bodenhamer, and is as follows: 1. Congenital narrowing of the rectum or anus without complete occlusion; 2. Complete occlusion of the anus by a membranous diaphragm or well-formed skin; 3. The anus is absent, and the rectum ends in a blind pouch at a point more or less distant from the perineum; 4. The anus is normal in appearance, but ends in a cul-de-sac, and the rectum ends in a blind pouch at a variable distance above this point; 5. The anus is absent, and the rectum ends by a fistula at any point of the perineum or sacral region; 6. The anus is absent, and the rectum ends in the vagina, the bladder, or the urethra; 7. The anus and rectum are normal, but the

ureter, vagina, or urethra opens into the rectal cavity; 8. The rectum is totally absent.

1. **CONGENITAL NARROWING OF THE RECTUM OR ANUS, WITHOUT COMPLETE OCCLUSION.**—This variety of malformation is probably more common than is generally supposed, as it escapes notice if the narrowing is not sufficient to produce marked symptoms of obstruction; and probably in many cases of this nature, in which the stenosis is not extreme, the efforts of the child in passing the fæces bring about the necessary amount of dilatation. As the stenosis may not be sufficient to prevent the escape of the semifluid fæces of infant life, the condition may not be detected for some time, and it is only as the child becomes older and the fæces become more consistent that accumulation takes place in the rectum and attention is directed to the malformation. (Fig. 1).

FIG. 1.



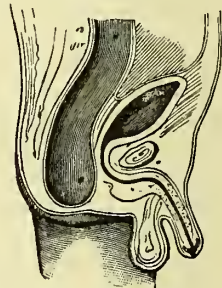
Congenital Narrowing of the Rectum and Anus.

Treatment.—The treatment of this variety of congenital stenosis is best conducted by gradual dilatation, which may be carried out by the daily introduction into the bowel of graduated bougies, or by the introduction of the oiled index finger of the mother or the nurse, which is by far the best of all bougies for this purpose.

2. **COMPLETE OCCLUSION OF THE ANUS BY MEMBRANOUS DIAPHRAGM OR BY WELL-FORMED SKIN.**—In this form of malformation closure of the anus may be caused by a diaphragm of mucous membrane or skin, which appears to be due to the adhesion or skinning over of the surface of the anus, the rest of the proctodæum being normally formed (Fig. 2).

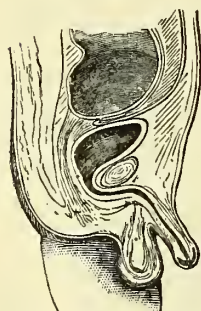
Treatment.—The treatment consists in making a crucial incision at the position of the anus, opening the rectal pouch, and evacuating the fæces and trimming off the edges of the mucous membrane and skin. The wound should be dusted with iodoform and dressed with a pad of antiseptic gauze, and the subsequent management of the case consists in keeping the anus well dilated for some time to prevent cicatricial contraction.

FIG. 2.



Complete Occlusion of the Anus by Membranous Diaphragm or Skin (after Ball).

FIG. 3.



The Anus is absent, and the Rectum ends in a Blind Pouch (after Ball).

3. **THE ANUS IS ABSENT, AND THE RECTUM ENDS IN A BLIND POUCH AT A POINT MORE OR LESS DISTANT FROM THE PERINEUM.**—In this variety of malformation the rectal pouch may terminate near the skin, or it may end high up in the pelvis and the space between it and the perineum be filled with cellular tissue, or in other cases a distinct fibrous cord can be traced from the termination of the rectum to the skin (Fig. 3).

Treatment.—In the treatment of this malformation—and, in fact, of all forms of imperforate rectum in which complete occlusion exists—the duty of the surgeon is very clear; he should, as soon as possible, attempt to reach the rectal pouch by a perineal incision. The earlier this is attempted the better, for delay in operating certainly conduces largely to a fatal result. I cannot subscribe to the opinion of those who advise delay until the rectum is distended with fæces and gas, which may make

the position of the rectal pouch more apparent, but which is not unattended with the risk of rupture of the intestine and exhaustion of the patient; and it has also been shown that by delay the meconium becomes reduced in bulk through the absorption of the fluids. It should be remembered that the rectum in infants descends in the hollow of the sacrum and is close to the bone, and except at its upper portion is uncovered by peritoneum posteriorly; in front its peritoneal investment descends to a much lower level, and its close relation in this aspect to the genito-urinary tract is an additional reason for the selection of the posterior region for exploratory operation. Various operative measures have been recommended and resorted to in cases of imperforate rectum.

Puncture with a Trocar Canula.—The introduction through the perineum of a trocar and canula was formerly advised, and by its use I have seen the rectum reached and meconium evacuated; but subsequently it is usually found necessary to enlarge the wound made by the instrument to secure free exit of fecal matter, so that the procedure possesses no advantage over the perineal incision, and has the disadvantage that the rectal pouch may be entirely missed by the trocar and important structures injured by its blind introduction.

Perineal Operation.—This is considered the best operation to undertake in these cases, since, if successful, it leaves the patient with an anus in the normal position, and often with fair control of the bowels, for the anal sphincter is frequently well developed in spite of the malformation of the rectum. In performing this operation the child should be placed in the lithotomy position, and the incision should be made in the median line of the perineum from the root of the scrotum to the coccyx. The tissues should be divided slowly, any bleeding vessels being secured as they are met with. The surgeon should explore the wound with the finger during the operation, to discover, if possible, the bulging of the rectal pouch, and should be careful to make the deepest incisions posteriorly. In a female infant the finger introduced into the vagina during the operation may give the surgeon some information as to the position of the rectum; or if the mass of fibrous tissue in which the rectum sometimes terminates is seen or felt, it may serve as a guide to the position of the rectal pouch. Nearness of the tuberosities of the ischium is a sign of absence of the rectum; and if it is found that the vagina or bladder fills up the concavity of the sacrum, it is an indication of a high termination of the rectal pouch. The incisions may be carried with safety to the depth of an inch and a half or two inches, and when the rectal pouch is reached it should be incised. After the meconium has escaped the wound in the rectum should be sufficiently enlarged, and, if possible, its edges should be brought down and sutured to the skin of the perineal wound, care being taken in passing the sutures and in introducing a drainage-tube to leave no pocket around the bowel for the accumulation of discharges. The suturing of the edges of the bowel to the skin is a most important procedure, and one which diminishes largely the amount of contraction in the newly-formed anus; it may, however, be found impossible to bring down the edges of the rectal wound to the skin in cases where the rectum terminates high up in the pelvis. In such cases a large flexible catheter or a metallic tube may be introduced and held in place by tapes; but it is difficult to keep it in position, as it is apt to be displaced by the straining efforts of the child. Verneuil has suggested excision of the coccyx in the early part of the operation, which facilitates the search for the gut, and in case it is found this procedure enables the surgeon more readily to attach the edges of the rectal pouch to the skin. The dressing of the wound should consist in dusting the parts with iodoform and applying a pad of antiseptic cotton, to be held in position by means of a T bandage.

When the surgeon has carried his dissection up into the pelvis as far as he considers it is safe, an inch and a half or two inches, and has failed to reach the rectal pouch, he should then consider the advisability of abandoning the attempt to reach the gut through the perineum, and should endeavor to open the intestine either in the left groin (Littre's operation), or in the left loin behind the peritoneum (Amussat's operation), or in the right groin (Huguier's operation). Of these operations, that in the left groin is to be recommended, as it opens the bowel near its natural termination. If the surgeon decides upon this operation, he should make an incision from one and a half to two inches in length, half an inch above and parallel with Poupart's ligament, beginning at a point opposite the junction of the middle with the outer third of this structure. Or an incision suggested by Ball, following the line of the linea semilunaris, stopping just short of Poupart's ligament, may be substituted for the former incision. The skin and muscular layers being cut through, the fascia transversalis and peritoneum may be pinched up together, and a small opening made in them, through which a director should be passed, and the two can then be divided with one incision. It is sometimes difficult to determine whether the bowel presenting in the wound is the small or large intestine; this can be ascertained by gently drawing out a coil: if it be the small intestine, it can be drawn out with ease, and the mesentery will show that it is not the portion of the bowel sought for, and it should be replaced. On the other hand, the large intestine cannot be so readily drawn out, and its mesocolon, if it have one, would be found attached to the left side. The bowel should next be secured to the edges of the wound by several sutures of fine silk or catgut, which should be introduced by passing a curved needle through the skin and parietal peritoneum near the edge of the wound, and then transfixing a portion of the bowel; after which the needle should be made to transfix the peritoneum and skin again, being brought out a short distance from the point of insertion; the stitches should then be secured. Sutures should be applied in this manner on each side and at the extremities of the incision, after which the gut should be incised to a sufficient extent and the meconium allowed to escape. After the escape of the latter the wound should be carefully cleansed, and the edges of the gut incision may be attached to the skin by a few silk sutures. The surgeon may introduce the finger or a flexible rubber catheter into the opening in the gut to ascertain, if possible, the point of termination of the rectal pouch; and if it is found to be near the upper portion of the perineal incision, he may deepen the latter on a guide introduced through the artificial anus. It has, however, been found better to rest satisfied with the relief afforded by colotomy, and to postpone for a time the attempt to form an anus in the perineal region, for the majority of cases in which this has been attempted have been followed by a fatal result. Attempts to accomplish this result some months after the performance of colotomy have been more satisfactory, as is seen in cases reported by Byrd and Krönlein. When the patient has attained some age, and an examination through the artificial anus in the left groin shows that the rectal pouch terminates well down in the pelvis, a director or rubber catheter may be introduced through the colotomy wound and made to enter the pouch, and project at the anus, if it be present, or at some point of the perineum. This may then be cut down upon as a guide, and the gut may be opened and sutured to the skin if the edges can be drawn down to that point.

If the surgeon should prefer to make an attempt to open the bowel in the left lumbar region, the best guide to the position of the colon is a line half an inch posterior to a point midway between the two superior spinous processes of the ilium; if he fails to find the large intestine, and distended small intestine

shows itself in the wound, it is better to open this and stitch it to the wound, rather than to abandon the case and allow the patient to perish by intestinal obstruction.

The results obtained by the various operations for the relief of the symptoms due to imperforate rectum show that, in point of safety and as a matter of comfort to the patient, the perineal operation is to be preferred. Cripps has collected 100 cases of the various operations for the relief of imperforate rectum; his table, although exhibiting a high rate of mortality, 50 per cent., shows that the largest number of recoveries followed the perineal operation, and the next in number were those cases in which the colon was opened in the iliac region. The expediency of an operation for the establishment of an artificial anus, either in the perineum or in the groin, in young children with imperforate rectum, is evidenced by a number of well-attested cases in which the patient lived for years afterward in comfort.

4. THE ANUS IS NORMAL IN APPEARANCE, BUT ENDS IN A CUL-DE-SAC, AND THE RECTUM ENDS IN A BLIND POUCH AT A VERY LITTLE DISTANCE ABOVE THIS POINT.—In this form the anus and rectum may be separated by a membranous partition of greater or less thickness, or a portion of the bowel may be impervious, or there may be multiple obstructions, or the anal portion may communicate with the vagina in the female and the rectum end in a cul-de-sac (Fig. 4). The variety of malformation in which the anus is normal, but is separated from the rectum by a membranous partition of greater or less thickness, is not uncommon. It is apt to escape notice for some time, as the anus is normal in appearance, and it is only when the nurse or mother notices that the child passes no fæces and the belly becomes swollen, or vomiting begins, that the nature of the trouble is suspected. The introduction of the finger or probe into the anus will soon reveal the nature of the trouble. An attempt should at once be made to reach the rectal pouch by an incision through the anus backward toward the coccyx, and if the gut be found it should be brought down and sutured to the edges of the anal wound. This procedure is much safer than puncture through the anus, which the surgeon might feel tempted to employ if the partition between the two cavities did not seem very thick. If it be found impossible, after a careful dissection in the perineal region, to find the rectal pouch, the surgeon should abandon this operation, and attempt to reach the gut by an incision in the left iliac region.

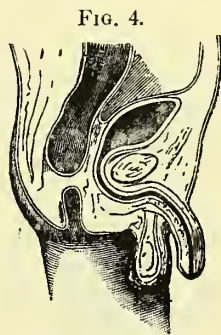


FIG. 4.
Anus ends in a Cul-de-sac, the Rectum ends in a Blind Pouch (after Mollière).

5. THE ANUS IS ABSENT, AND THE RECTUM ENDS BY A FISTULA AT ANY POINT OF THE PERINEUM OR SACRAL REGION.—The rectum may open at some portion of the perineum or sacral region, or it may terminate in a narrow channel under the raphé of the perineum and open at the prepuce or at the symphysis pubis, or may end in several fistulæ at different points (Fig. 5). Such patients may have satisfactory evacuations through the fistulæ, and may live for months or years without suffering much inconvenience from the deformity.

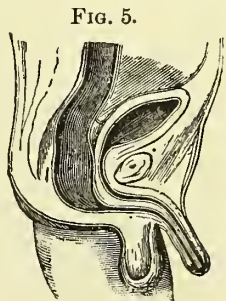


FIG. 5.
Anus is absent; Rectum ends by a fistula at the prepuce (after Ball).

Treatment.—If a child so suffering shows evidence of discomfort by reason of the fæces not passing sufficiently freely through the fistula, this should first be dilated or increased in size by incision, and if relief

from the symptoms be obtained, no further operation should be attempted as long as the child remains in comfort. When the patient has attained an age when a more radical operation can be undertaken, the fistula may be explored with a probe or director, and the position of the rectal pouch ascertained if possible; if it be in a favorable position, a perineal opening should be made to reach the rectum, and when it has been found the bowel should be opened and its edges brought down and sutured to the edges of the perineal wound. The fistulous tract should be laid open or touched with the actual cautery, and allowed to heal by granulation.

6. THE ANUS IS ABSENT, AND THE RECTUM ENDS IN THE VAGINA, BLADDER, OR URETHRA.—These malformations, according to the point of termination of the rectum, are classified as *atresia ani vaginalis*, *atresia ani vesicalis*, and *atresia ani urethralis*. Leichtenstern's statistics show that 40 per cent. of rectal malformations are of this nature. This tendency of the rectum to terminate in the genito-urinary tract is remarkable when we consider the definite separation which exists between the rectum and the genito-urinary tract in the adult: it is attributed by Ball to the method of development of the proctodæum, or a tendency to reversion to the cloacal type of birds and lower animals.

Atresia Ani Vaginalis.—In this form the rectum terminates in the posterior walls of the vagina, either by a small or large aperture. The opening may be situated immediately within the fourchette, or may be located high up in the canal (Fig. 6). If the rectal opening is sufficiently large, the patient does not exhibit any symptoms of intestinal obstruction, and the nature of the deformity is only ascertained upon inspection of the parts, when it is found that the anus is absent, and that fecal matter escapes from the vulva.

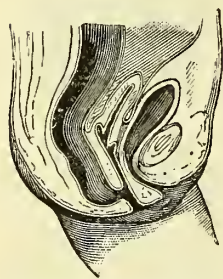


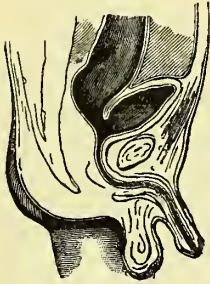
FIG. 6.
Anus is absent; Rectum ends in the vagina. (After Ball.)

Treatment.—If the patient suffers no inconvenience, operative treatment may be postponed until she has attained some age, when the greater development of the parts will conduce to a favorable result. Operations for the relief of this variety are the most satisfactory in their results of all those that have been devised for the cure of congenital malformations of the rectum. When an operation is decided upon, the one which is followed by the best results is performed in the following manner: A director is passed through the vaginal opening into the rectum and is pushed backward, its point being made to project as near as possible to the normal position of the anus; this is cut down upon from the perineum and the rectum is exposed and incised. The rectal wound being then sufficiently enlarged, the gut is dissected loose and its edges are brought down and secured to the skin by sutures. By this dissection of the rectum and bringing down of its edges, the opening into the vagina, if it be a low one, is obliterated. If a high opening into the vagina remain after the anus has been established in its normal position, an operation may be undertaken later to close this recto-vaginal fistula. An ingenious operation, devised by Rizzoli, for the relief of this malformation is performed as follows: An incision is carried from the lower margin of the vaginal anus backward through the perineum toward the coccyx, care being taken not to open the intestine; the termination of the rectum with its vaginal orifice is now carefully dissected out, and the abnormal anus is transplanted to its natural situation and secured in that position by a few sutures, after which the perineal and vaginal wounds are brought together by deep sutures.

Atresia Ani Vesicalis.—In this variety the rectum communicates with the

bladder, either by a narrow orifice near the base of the organ or by an opening near its fundus (Fig. 7). The absence of the anus and the escape of faecal matter intimately mixed with urine at the time of urination would point to the nature of this very serious malformation.

FIG. 7.



Anus absent; the Rectum ends in the Bladder. (After Ball.)

Treatment.—In the treatment a staff may be introduced through the urethra into the bladder, and an incision made through the perineum into the neck of the bladder, as in lithotomy, and continued into the rectum. As the result of this operation the immediate symptoms of obstruction may be relieved, but the patient is left with a urinary and faecal fistula. Ball suggests a laparo-colotomy, and, when the colon has been found, its complete division, with closure of the lower portion and the bringing out of the upper portion at the wound, and securing it in that position to establish an artificial anus. This

operation, although attended with greater immediate risk, has the advantage of leaving the patient with control over his urinary excretion.

Atresia Ani Urethralis.—In this form the rectum communicates with some portion of the urethra, allowing the escape of a small amount of faecal matter, which passes more or less in the intervals between urination. The urethral opening is usually so small that faeces cannot escape in sufficient quantity, and the symptoms of intestinal obstruction are soon developed (Fig. 8).

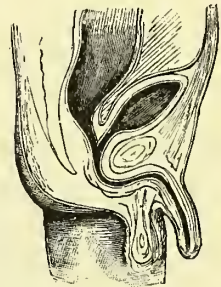
Treatment.—The treatment consists in attempting to find the rectum by means of perineal incision, opening it, and bringing down the edges of the gut and suturing them to the skin.

7. THE ANUS AND RECTUM ARE NORMAL, BUT THE URETERS, VAGINA, AND UTERUS OPEN INTO THE RECTAL CAVITY.—As this is a malformation in which occlusion of the bowel does not exist and life is not endangered by its presence, no immediate operation is called for. Where the ureters open into the rectum, no operative interference could be of any avail, but in that form in which the vagina or uterus opens into the rectum, and the child has attained some age, an operation to close the fistula and replace the organs may be attempted.

8. THE RECTUM IS TOTALLY ABSENT.—This differs from the third variety of malformation only in the amount of rectum which is wanting, and its existence may be suspected in those cases in which an exploration of the pelvis by perineal incision fails to reveal the presence of the rectal pouch. This condition is to be treated by laparo-colotomy, in the left inguinal region, and the formation of an artificial anus.

9. THE LARGE INTESTINE IS TOTALLY ABSENT.—This condition is often associated with a faecal fistula at the umbilicus or some other portion of the body, and its treatment consists in securing a free exit of faeces from this fistula by dilatation or careful incision, or by the formation of an artificial anus if no fistula be present.

FIG. 8.



The Anus is absent; the Rectum ends in the Urethra. (After Ball.)

III. DISEASES OF THE ANUS.

PRURITUS ANI.

This affection is occasionally seen in childhood, and is characterized by a painful itching in the region of the anus, which causes the child constantly to scratch the part, so that the skin in the vicinity becomes thickened, eczematous, and moist from exudation as a result of the constant irritation. Pruritus ani may result from various causes—from the presence of oxyuris vermicularis in the rectum, from eczema of the anus, from pediculi or scabies, or from the presence of a vegetable parasite, as is the case in eczema marginatum. In other cases in which the itching is not attributable to any of the above-named causes it can often be traced to improper diet or chronic constipation.

Treatment.—Where the condition can be traced to the presence of eczema, the parts should be frequently bathed with hot water and washed carefully with green soap, and one of the following lotions may be used:

R_y. Acidi carbolici ℥xx.
Liquor. calcis f℥vj.—M.

Or,

R_y. Acidi carbolici f℥ss.
Glycerini f℥j.
Aquæ q. s. ad f℥vj.—M.

Or the following ointment may be applied:

R_y. Ung. picis liquidæ ʒj.
Ung. zinci oxidi ʒiij.
Ung. aquæ rosæ ʒiv.—M.

When the itching can be traced to the presence of parasites, either animal or vegetable, the use of some of the antiparasitic lotions or ointments appropriate for the individual case will rapidly effect a cure. Where the condition is dependent upon errors in diet, a change of diet will often be followed by satisfactory results. Where the trouble arises from chronic constipation, a change of diet should be made and laxatives should be administered, or enemata or suppositories of glycerin should be employed.

SYPHILITIC AFFECTIONS OF THE ANUS.

Mucous patches and moist papules occur with comparative frequency in the region of the anus as the result of congenital syphilis. Allingham speaks of numerous cracks or fissures of the mucous membrane of the anus in children suffering from hereditary syphilis. Condylomata may appear upon these syphilitic lesions: they are acuminate and spring from previously existing papules or mucous patches, and are accompanied by discharges of a characteristic fetid odor. These growths are to be distinguished from the simple forms of vegetation which frequently occur in this region, and are not dependent upon the presence of inherited syphilis.

Treatment.—The treatment should be both constitutional and local. The constitutional effects of mercury can best be obtained in young children by the use of a binder spread with mercurial ointment applied around the abdomen. The local treatment of the anal lesions consists in the application of the

solid stick of nitrate of silver, or, better, the acid nitrate of mercury, or in dusting them with a powder consisting of equal parts of calomel and oxide of zinc.

VEGETATIONS OR WARTS OF THE ANUS.

Vegetations of the anus are not infrequent in childhood, and the growths may attain great size. They are similar in structure to warts situated in other parts of the body, and are papillary overgrowths covered with squamous epithelium. From their situation they are apt to be kept in a moist condition, and as a result there is often present a certain amount of offensive discharge.

Treatment.—If the parts can be kept perfectly dry, a cure will usually rapidly result: with this end in view, when the growths are not large, dusting with lycopodium or powdered oxide of zinc will often be followed by their disappearance. If the growths are large, they may be touched with the solid stick of nitrate of silver or a saturated solution of chromic acid; or they may be destroyed by the application of the actual cautery, or trimmed away with scissors. The objection to the latter mode of removing them is the profuse hæmorrhage which may result, but this can generally be controlled by the application of a firm compress to the bleeding surface.

FISTULA IN ANO.

Fistula in ano is an affection in which there is a communication between the mucous surface of the rectum or anus and the skin in its immediate neighborhood. A complete rectal fistula is one in which there is a sinus leading from the rectum to some point of the skin in the region of the anus; an incomplete fistula or an internal rectal sinus is one in which there is a sinus passing from the rectum into the perirectal cellular tissue; another form of incomplete fistula is known as the external rectal sinus, and is one in which there is an opening on the skin passing into the cellular tissue around the rectum, but not perforating the wall of the gut. Fistula in ano is certainly a rare affection in infants and children. Allingham mentions the fact of its occurrence in children of a very tender age. I have myself seen a few cases in children, and recall a case of complete fistula in a child a few months of age. The affection may result from perforating ulceration of the mucous membrane of the rectum, or from an ischio-rectal abscess opening into the rectum or through the skin in the vicinity of the anus, and also from wounds involving the rectum or anus.

Diagnosis.—This affection is usually not difficult to diagnose if the finger be introduced into the rectum and a probe passed into the external opening, when, by a little careful manipulation, the probe may be made to enter the bowel if the fistula be a complete one. In the incomplete form of fistula known as internal rectal sinus, careful palpation of the tissues surrounding the anus will often reveal an indurated mass of tissue which indicates the position of the internal fistula, and the finger introduced into the rectum may also feel the orifice of the internal opening; while the discharge of pus with the stool points to the existence of this affection. In the form of incomplete fistula known as external rectal sinus, if the finger be introduced into the rectum and a probe passed into the external opening, it can be felt at some point to come near the wall of the bowel. In children it should be remembered that, in certain cases of disease of the bones of the spine, of the sacrum, or of the pelvis, the purulent matter passing through the connective tissue about the rectum may find its way to the surface and perforate the skin in the neighborhood of the anus; or it may open into the rectum and escape by the

anus. A careful examination of the patient, however, will reveal the origin of the pus and show that it is not a case of ordinary fistula in ano.

Treatment.—The treatment of this affection consists in the free division of all the tissues between the internal and external opening of the fistula, and is accomplished as follows: A director having been passed into the external opening of the fistula, the finger is introduced into the rectum, and when the point of the director is felt it is passed through the internal opening and brought out at the anus; the superimposed tissues are then divided with a bistoury. The track of the fistula should next be carefully explored to discover the presence of any branching sinuses running off from it, and if these be found they should be freely laid open. The wound resulting should next be touched with the solid stick of nitrate of silver, or curetted, irrigated with a solution of bichloride of mercury, and packed with strips of iodoform gauze; this dressing should be changed at intervals of a few days, and the wound is to be allowed to heal by granulation. In cases of incomplete external fistula the director should be introduced into the external opening, and where its point comes in contact with the gut, guided by the finger in the rectum, it should be made to perforate, and its point brought out at the anus. The superimposed tissues are then divided, as in the operation for complete fistula. In the variety of incomplete fistula known as internal rectal sinus, the position of the fistula being located as before described, an incision should be made through the skin at this point, and a director introduced and made to enter the rectum, its end being brought out of the anus. The subsequent treatment of the case differs in no wise from that of the complete fistula. Another method of treatment in incomplete fistula of either variety is to lay the sinus freely open down to the bowel without dividing the sphincter, and to pack the wound with iodoform gauze: in this way a cure may often be brought about. In any case of fistula in which the internal communication is very high up in the rectum, and its division by the knife is considered unsafe by reason of the hæmorrhage which may result, an elastic ligature may be introduced through the external opening by means of an eyed probe and brought out at the anus, after which the ligature is tied and allowed to cut its way out, and the wound resulting is treated like that following division of the tissues by the knife.

FISSURE OF THE ANUS.

Fissure of the anus is an affection in which there exists at some portion of the mucous membrane of the anus a small linear ulcer, which causes great pain at stool or after the bowels have been moved. This affection is considered infrequent in childhood, but I am of the opinion that its presence is not so unusual as is generally supposed, and feel sure that a careful inspection of the anal region in children who complain of pain at or after stool will often show its presence. Allingham and Curling mention cases which they have met with in quite young patients, and I have myself seen cases of this affection in children. Kjellberg of Stockholm among 9098 children found 128 cases of fissure of the anus. The majority of these children were less than one year of age, and in 73 cases the patients were less than four months old. Jacobi thinks fissure of the anus a much more common affection in children than is generally supposed, and believes that many of the fretful children who sleep badly and cry constantly, and often present symptoms similar to those of vesical calculus, suffer from fissure of the anus.

Diagnosis.—Fissure of the anus should be suspected in cases where pain is experienced during or after stool and where the stool contains a few drops of

blood. In such cases a careful inspection of the part will usually reveal the presence of a fissure. The rectum should at the same time be examined with the finger for the presence of polypus, which frequently coexists with fissure of the anus.

Treatment.—The treatment of this affection in children can generally be successfully accomplished by an application of a 20-grain solution of nitrate of silver to the ulcer, or by lightly touching the surface with the solid stick of nitrate of silver, and afterward keeping the parts well covered with an ointment composed of thirty grains of iodoform or aristol to the ounce of vaseline, the bowels being kept in a soluble condition. In cases which are found intractable division or stretching of the sphincter may be resorted to.

STRICTURE OF THE ANUS.

This affection may be congenital or may result from an operation in the vicinity of the anus. The treatment of stricture of the anus consists largely in gradual dilatation of the contracted orifice, either instrumental or digital; if this fails to relieve the condition, a careful incision of the contracted parts should be practised, and subsequent dilatation should be employed for some time.

MARGINAL ABSCESS.

This affection consists in circumscribed suppuration starting in the mucous follicles of the anus, or from a fissure of the anal margin, and is a much more common and less serious affection in childhood than ischio-rectal abscess. Although painful, it is not apt to result in the formation of a fistula in ano.

The treatment consists in making a free opening with a bistoury, and to accomplish this the tip of the finger should be passed into the rectum to steady the abscess-cavity and make it more prominent before it is incised; the wound should then be dressed with iodoform gauze or with lint saturated with carbolized oil, and usually heals promptly.

DIPHTHERIA OF THE ANUS.

This affection is occasionally seen in children suffering from diphtheria of the pharynx, and usually develops late in the disease and in cases in which the system has been profoundly impressed. The deposit of diphtheritic membrane may involve the anus and extend on to the buttocks, and to the mucous membrane of the vulva in female children.

The prognosis is extremely unfavorable, and the cases which have come under my personal observation have all terminated fatally in spite of treatment.

The treatment consists in the employment of such constitutional remedies as are appropriate for diphtheria, and the local application to the affected surface of a solution of bichloride of mercury, 1 : 2000 or 1 : 4000, followed by the use of an ointment of iodoform.

IV. DISEASES OF THE RECTUM.

PROCTITIS.

Inflammation of the rectum, or proctitis, is an affection frequently seen in childhood. It may result from injury to the mucous membrane by the fæces or by materials contained in the fæces, or it may follow from traumatism received

from without. It is recognized in two forms—acute catarrhal and chronic catarrhal proctitis.

Acute Catarrhal Proctitis.—In this affection the inflammatory symptoms are limited to the rectum, and the disease is characterized by great tenesmus and the frequent passing of bloody mucus, at first mixed with feces. In addition to these symptoms there are usually present œdema of the mucous membrane of the anus and of the lower portion of the rectum, and vesical irritation; and as a result of this condition and the constant straining there is often observed a partial prolapsus of the rectum. Many of the symptoms presented are those of acute dysentery, but the abdominal pain and the constitutional features of the latter affection are generally wanting.

Treatment.—The patient should be kept in a recumbent posture, and small doses of castor oil or one of the saline cathartics, either sulphate of sodium or of magnesium, or one of the natural mineral waters, should be administered to secure free evacuation of the bowels. The diet should be restricted to milk, animal broth, and eggs. If, after the bowels have moved, tenesmus continues, an enema consisting of a few drops of tincture of opium and starch-water should be injected into the rectum; or a rectal suppository containing powdered opium grain $\frac{1}{8}$, extract of belladonna grain $\frac{1}{6}$, iodoform grain 1, should be administered, and if the patient shows signs of exhaustion stimulants should be employed. The disease is usually of short duration, and under treatment the symptoms generally subside in a few days.

Chronic Catarrhal Proctitis.—This disease usually results from the acute affection, and is characterized by the absence of pain and tenesmus, although in some cases in which ulceration of the mucous membrane of the rectum exists there may be discharged a small quantity of blood and muco-purulent matter.

Treatment.—The diet should be regulated as in acute proctitis, and if the evacuations are not sufficiently free the bowels should be moved by the administration of a saline cathartic. The local treatment should consist in the use of enemata of nitrate of silver, $\frac{1}{4}$ to 1 grain to an ounce of water, which should be gradually increased in strength until they begin to cause pain; suppositories of iodoform and extract of belladonna may also be employed with advantage.

PERIPROCTITIS.

Periproctitis is an inflammatory condition involving the connective tissue surrounding the rectum. It may result from septic causes or direct injury, or may arise from the introduction of foreign matter through ulceration or perforation of the rectum. Abscess or gangrene of the cellular tissue may result, with subsequent involvement of the skin; erysipelas also may attack this region, giving rise to erysipelatous periproctitis.

Treatment.—In this affection, as soon as the swelling and induration can be detected, free incision should be made through the skin and into the cellular tissue outside of the margin of the anus, and the wounds thus produced irrigated with a solution of bichloride of mercury, 1:1000 or 1:4000, or touched with a solution of chloride of zinc, 15 grains to the ounce. The surfaces then should be dusted with iodoform, and covered with a bichloride-gauze and cotton dressing. If the parts are found to be gangrenous, a charcoal or an antiseptic poultice should be applied, and the patient should be given alcohol and tonics with a liberal diet.

ISCHIO-RECTAL ABSCESS.

Ischio-rectal abscess consists of a purulent collection in the loose cellular tissue surrounding the rectum. It is a most painful and serious affection, and

is the most frequent cause of fistula in ano. It may arise from injuries of the rectum, either from within or from without, from phlebitis or periphlebitis of the hæmorrhoidal veins, or from the escape of fæcal matter into the cellular tissue through ulcers perforating the rectum. The most characteristic symptom of ischio-rectal abscess is a sense of fulness in the lower portion of the rectum, with throbbing pain, which is increased at the time of stool. Where this affection is suspected a careful examination of the rectum with the finger will often disclose a bulging of the rectal wall at some point, and this is often accompanied by swelling and œdema of the skin near the anus; the presence of fluctuation in this region will often be revealed upon palpation.

Treatment.—This form of abscess demands prompt and free opening, and by this treatment alone is the pain relieved and the risk of the formation of a fistula in ano avoided. In opening these abscesses I usually follow the practice of Allingham, who recommends that the patient should be etherized and placed in the lithotomy position. An incision should be made at a little distance from the anus parallel with the sphincter, the abscess-cavity laid freely open, and the finger introduced into the wound to break down any secondary cavities or loculi. If it is found that there has been much undermining of the tissues, incisions should be made at right angles to lay all cavities freely open. The abscess-cavity should then be irrigated with a solution of bichloride of mercury, 1 : 2000 or 1 : 4000, or with a 1 : 40 solution of carbolic acid; and the wound should next be carefully packed with lint saturated with carbolized oil, 1 : 30, or, as I prefer, with iodoform gauze. An external dressing of iodoform or bichloride gauze and a pad of bichloride cotton is then applied to the wound and held in position by a T bandage. This dressing need not be disturbed, unless it become loose or soiled, for several days, when the cavity should be irrigated and a few strips of gauze laid lightly in it. The wound should be allowed to heal by granulation. If the bowels do not move in one or two days, a gentle laxative may be administered. By this method of treatment the cavity of the abscess rapidly heals, and a cure results without the formation of a fistula in ano.

ULCERATION OF THE RECTUM.

Ulceration of the rectum is not a common affection in childhood, but it sometimes results from chronic dysentery or chronic catarrhal proctitis.

The treatment consists in the local use of injections of nitrate of silver, grain $\frac{1}{4}$ to 1 to the ounce of water, and in the use of suppositories of iodoform. A restricted diet should also be enjoined, and the bowels should be regulated.

STRICTURE OF THE RECTUM.

This affection may result from the presence of new growths, from the contraction following wounds of this organ, the result either of accident or operation, and also from congenital malformations of the rectum; inherited syphilis is mentioned as occasionally causing congenital stricture of the rectum.

The treatment consists in gradual dilatation of the rectum, either instrumental or digital; if the condition be due to the presence of growths, their removal should be accomplished if possible; and if due to inherited syphilis, antisyphilitic treatment is indicated, in addition to the local measures.

SYPHILIS OF THE RECTUM.

Lesions of the rectum, due to inherited syphilis, are occasionally seen in childhood. A case of gummatous infiltration of the coats of the rectum in a child ten years of age, at the same time exhibiting well-marked symptoms of

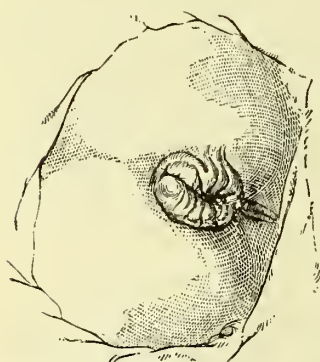
inherited syphilis, has been described by Ball; and Oser of Cracow has reported two cases of gummatous infiltration of the intestines in children suffering from congenital syphilis.

The treatment of syphilitic lesions of the rectum consists in the administration of mercury or iodide of potassium, as in the treatment of corresponding syphilitic lesions in other parts of the body.

PROLAPSUS OF THE RECTUM.

Prolapsus of the rectum consists in the protrusion of a portion of the rectum through the anus, and occurs in three varieties: 1. A portion of mucous membrane protrudes from the anus (partial prolapsus); 2. The entire thickness of the walls of the rectum is included in the prolapse (complete prolapsus); 3. There exists an invagination as well as a prolapsus of the rectum. This affection in some one of its varieties is very common in childhood, and the frequency of its occurrence may be accounted for on both anatomical and pathological grounds. The looseness of the attachment of the submucous connective tissues of the walls of the rectum is a well-recognized anatomical fact; and this condition is an important factor in the production of prolapsus. The straightness of the coccyx in children is also said to favor its production. In infants and young children the great amount of straining that seems to be required to bring about satisfactory evacuations is also productive of this affection; this straining

FIG. 9.



Prolapsus of the Rectum. (After Bryant.)

has been ascribed by Jacobi to the anatomical fact that in children it is not uncommon to find two or three angular flexures in the lower part of the colon. The habit so common with mothers and nurses of placing children upon the chamber utensil and allowing them to spend a large portion of time in that position is certainly, to my mind, a frequent cause of the development of prolapsus, and is a custom which cannot be too severely condemned. In many cases the constant straining due to the presence of vesical calculus or rectal polypus, or to a contracted prepuce, may be an important factor in the production of this affection. Improper diet, or the custom of allowing children to eat at all hours during the day—and as a result of this over-feeding the pro-

duction of a large number of passages—may also be mentioned as a cause. That improper diet and over-feeding produce prolapsus of the rectum is, in my mind, very clearly proved by the fact that at the Children's Hospital of Philadelphia we often have children admitted to the wards for operation who have suffered from this affection for months: under the use of tonics, proper diet, and regulation of the bowels they fail to further present prolapsus, and are thus soon relieved of the condition without operative interference. Mr. Holmes of London makes a similar observation as to his experience in this affection.

Symptoms.—The characteristic symptom is the protrusion, during defecation, of a reddish-purple mass covered with mucous membrane: it is unaccompanied by pain, and usually undergoes spontaneous reduction as soon as the straining efforts cease. In the partial variety of prolapsus of the rectum little inconvenience is experienced, unless the prolapsed portion of the bowel is allowed to remain out for some time, when it may become congested or ulcerated; the latter condition is more likely to occur in cases of complete pro-

lapsus. When the prolapsus is of the third variety and is accompanied by invagination of the rectum, the symptoms of obstruction of the bowel exist, and gangrene of the protruded mass may occur. Death has resulted in such cases from obstruction as well as from peritonitis.

Diagnosis.—Prolapsus of the rectum is likely to be confounded only with hæmorrhoids, which is an extremely rare affection in childhood, or with polypus of the rectum. The appearance of the prolapse is very characteristic: the annular fold of tissue surrounding the whole anus with its depressed central orifice, and the fact that after reduction of the mass no tumor can be found in the rectum, would exclude the presence of polypus. The cases most likely to give rise to error are those of intussusception in children where the intussusceptum protrudes from the rectum, and resembles in appearance a prolapsus. Such cases have been found with prolapsus of the rectum; but if the surgeon makes a careful examination of the protruded mass, and takes into consideration the previous history of the case, such as sudden pain and collapse or the occurrence of more or less obstruction of the bowels, with the passing of blood and mucus preceding the appearance of the tumor through the anus, he will not be likely to confound the two affections.

Treatment.—The palliative treatment of this condition consists in returning the mass through the anus as soon as possible. This is best accomplished by placing the patient across the knees and making gentle pressure with the fingers over the whole mass of the tumor for a few moments, to return the contents of the bowels and the fluids effused in the tunics, and then pushing up the central portion first with the finger. Little difficulty is experienced in effecting this reduction in recent cases, but where the prolapsus has been down for some time and inflammatory effusion has taken place, it may be necessary to administer an anæsthetic before the reduction can be satisfactorily accomplished. The preventive treatment consists in not allowing the child to make prolonged straining efforts on the chamber-utensil or to have the bowels moved in a sitting posture. A child who is subject to prolapsus of the rectum should have the bowels moved while in the recumbent position on the bed-pan, or on the side, or in a standing posture; and the nurse should support the perineum and anus by two fingers placed one on each side of the anus, or by forcibly drawing the skin of the buttock to one side while the child is passing the stool. When the affection depends upon the presence of a vesical calculus, a contracted prepuce, or a rectal polypus or parasites, producing great straining efforts, the removal of the cause will usually effect a cure promptly. The importance of carefully regulating the diet has been previously mentioned, and care in this respect alone may bring about a cure. Enemata of astringent solutions, such as decoction of oak bark, a solution of alum, or cold water, or suppositories containing extract of *nux vomica* and ergot, have been employed; and of these the enemata of decoction of oak bark, or of cold water, are most satisfactory. In cases where these various palliative measures have failed to relieve the condition, I think the safest and, in my experience, the surest method of treatment is that recommended by Allingham. This consists in the application of nitric acid to the mucous membrane of the protruded gut. The child's bowels having been previously opened by the administration of a small dose of castor oil or by the use of an enema, he is etherized, and the surface of the prolapsed bowel is carefully cleansed and dried of mucus by wiping it with absorbent cotton; the whole surface of the mucous membrane is next painted with nitric acid applied with a swab, care being taken not to allow the acid to come in contact with the adjacent skin. A pledget of oiled cotton or lint is next introduced into the central depression

of the prolapsed mass, and by pressing it upward with the finger the mass is reduced. Finally, a pad is placed over the anus and held in position by bringing the buttocks together over it by means of broad strips of adhesive plaster. The bowels should be kept quiet for two or three days by the administration of a small amount of opium; and at the end of that time they should be opened by a laxative. The introduction of the oiled cotton or lint I have found in practice unnecessary, as it is apt to be passed by straining when the patient recovers from the anæsthetic; hence I generally omit its use, and merely coat the cauterized surface of the bowel with olive oil or vaseline before reducing it. The recurrence of the prolapsus may take place with the first few passages, but a permanent cure generally results from one application of the nitric acid. Should this, however, not be the case, cauterization may be repeated in a few weeks. The ligature and the clamp and cautery or actual cautery have been employed in the treatment of this affection, but as their use is attended with danger in cases of complete prolapsus of the rectum, and as I have never seen a case in a child in which the simpler and safer procedure, cauterization by nitric acid, has failed to give satisfactory results, I do not think their employment is to be recommended. In cases of prolapsus of the rectum in which invagination has occurred and the patient is suffering from obstruction of the bowels, if the mass cannot be returned under either an artificial anus should be made in the left inguinal region; and if the child survives after the invaginated portion of the gut has been removed by sloughing or other means, an attempt may be made to close the fæcal fistula in the inguinal region, and thus allow the fæces to escape through their natural channel.

HÆMORRHOIDS.

Hæmorrhoids are vascular tumors which occupy the lower portion of the rectum, and arise from dilatation or proliferation of the blood-vessels. They may be either internal or external, and are covered either by mucous membrane or skin. Hæmorrhoids are uncommon in childhood, but are occasionally seen, and may consist either of dilated veins or well-marked venous tumors. Allingham records a case of well-marked hæmorrhoids which he saw in a child three years of age. I have myself seen several cases in quite young children, and have seen recently with Dr. Starr a child three years of age who suffered from well-marked venous hæmorrhoids, which protruded and bled at stool, and presented symptoms severe enough to call for operative interference. Ball also has observed several cases in young children. The symptoms presented by hæmorrhoids in children are similar to those in adults, and consist in protrusion of the tumors and bleeding at the time of defecation.

Treatment.—As hæmorrhoids are apt to occur in strumous children, the administration of iron and cod-liver oil is often followed by decided benefit, and locally the use of astringent ointments and the regulation of the action of the bowels may be followed by marked amelioration in the condition. If, however, the tumors continue to bleed and to be protruded at stool, operative treatment is indicated, and the masses may be removed either by the use of the ligature or, as I prefer, by the clamp and cautery.

POLYPUS OF THE RECTUM.

Polypus of the rectum is a much more common disease in childhood than hæmorrhoids, and is characterized by the presence of a follicular tumor springing from the mucous membrane of the rectum at a point an inch or an inch and a half above the anus; it is attached by a pedicle. The form of

polypus most commonly seen in childhood is of the follicular or adenoid variety, and resembles in structure the normal mucous membrane of the rectum, from which it originates; but fibrous and cystic polypi have also been observed. Mr. Thomas Smith has recorded three cases of disseminated polypi of the adenoid variety occurring in young persons, and Cripps also reports cases of multiple polypi springing from the surface of the rectum and colon.

A rectal polypus is of a bright-red color when first extruded, but becomes darker and more venous in appearance after it has been protruded for some time and its circulation has been interfered with by constriction of the sphincter. The growths may be either single or multiple, and have pedicles varying from $\frac{1}{2}$ to 2 or 3 inches in length. Polypus of the rectum is comparatively rare in children: Bokai found 25 cases of this growth in 65,970 patients, and Jacobi says that he sees from 1 to 3 cases annually among 500 children. A rectal polypus is apt to produce expulsive efforts with tenesmus, and give rise to a sense of fulness or distress in the lower part of the rectum, and to be accompanied by the escape of glairy or bloody mucus or of blood.

Diagnosis.—The diagnosis is usually not difficult, as the growth is apt to present at the anus or to protrude from it during defecation, and a careful examination with the finger will disclose the presence of a pedicle to which the growth is attached. Polypus of the rectum is likely to be confounded with hæmorrhoids or prolapsus of the rectum, but a careful inspection and examination of the parts will disclose the nature of the trouble. Before examining a case of suspected polypus of the rectum it is well to give an enema, and when this is passed the growth is apt to be brought to the lower portion of the rectum or may present at the anus. In examining for polypus it is well to introduce the finger as far as possible into the rectum, and, as it is withdrawn, to make the examination of the walls with a sweeping motion, by which manipulation the pedicle of the polypus may be hooked upon the finger.

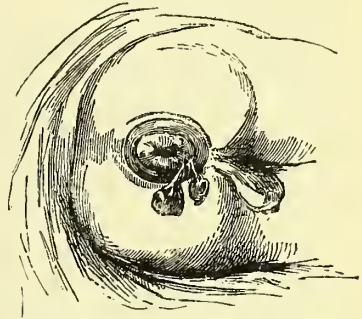
Treatment.—A polypus of the rectum may be seized with the fingers or forceps and twisted off, and the stump may be touched with nitrate of silver or with nitric acid; but I think the better method of treatment is to grasp the polypus and draw it out of the anus, so as to expose its pedicle, and to surround this with a ligature close to the mucous membrane, care being taken not to make sufficient traction to invert the wall of the rectum, which might thus be included in the grasp of the ligature. The ligature should next be firmly tied, and the tumor removed by dividing the pedicle in advance of the ligature. If a number of polypi exist, the same procedure should be repeated for each growth.

ANGEIOMA OR NÆVUS OF THE RECTUM.

This is also a rare affection. Mr. Howard Marsh has reported the case of a girl ten years of age who suffered from rectal hæmorrhage, in whom an examination revealed a nævoid growth in the lower portion of the rectum; and Mr. Barker has also published a case of this nature.

Treatment.—The treatment of nævus of the rectum consists in the use of

FIG. 10.



Polypi of Rectum with Prolapsus.
(After Ball.)

the ligature to strangulate the growth, or the application of nitric acid or Paque-lin's cautery.

MALIGNANT DISEASE OF THE RECTUM.

Malignant disease of the rectum is very rarely met with in childhood, but may occur either in the form of cylindrical-celled carcinoma or of sarcoma. Allingham, Quain, Cripps, and other observers have reported a few cases occurring in childhood.

Treatment.—The treatment consists in the excision of the growth if its situation be favorable for such a procedure; or linear rectotomy, which consists in freely dividing the growth together with the lower portion of the rectum, including the sphincter, may be practised with benefit, if obstructive symptoms are present. If the growth involves the high portion of the rectum and excision is not possible, colotomy should be performed.

WOUNDS OF THE RECTUM.

Wounds of the rectum may be caused by substances which reach the rectum through the alimentary canal, or by bodies introduced through the perineum or the anus; these wounds may be lacerated, incised, or punctured. Lacerated or punctured wounds may result from patients falling upon sharp bodies which enter through the perineum or anus, or from fragments of broken bones of the pelvis, causing in many cases extensive laceration of the parts about the rectum as well as of the rectum itself; they may be complicated by injuries of the bladder, vagina, or peritoneum. Lacerated wounds of the rectum may also result from the careless or forcible introduction of the nozzle of an enema-syringe; and laceration of this organ in children who have been subjected to unnatural intercourse should also be mentioned. Incised wounds of the rectum may result from operations upon this organ or from its accidental incision in the operation of lithotomy.

Treatment.—The treatment of incised or external lacerated wounds which involve only the lower portion of the rectum consists in controlling bleeding by the application of ligatures to the bleeding vessels; in washing the wound thoroughly with a solution of bichloride of mercury, 1:4000; in dusting the wound with powdered iodoform; and in providing for the escape of discharge by the introduction of a drainage-tube or catgut drain, and in bringing the edges together with catgut sutures. A gauze dressing should then be applied, and the bowels kept quiet for a few days.

In punctured or internal lacerated wounds of the rectum which do not extend high enough to involve the bladder or peritoneum it is better, in order to secure free drainage, to convert the internal punctured or lacerated wound into an open wound by the division of all the tissues, including the external sphincter and the skin. The wounds should then be washed with a solution of bichloride of mercury, packed lightly with iodoform gauze, and allowed to heal by granulation, the dressing being changed as often as it becomes soiled.

In a case of lacerated wound of the rectum complicated by wound of the bladder, perineal cystotomy should be performed to provide for the free escape of urine, and free drainage secured by division of the anal sphincter and the introduction of drainage-tubes if necessary. If a punctured wound of the rectum involves the peritoneum, with injury to the contained viscera, laparotomy should be performed, the wounds of the viscera should be sutured, and the peritoneal cavity irrigated, drained, and closed.

FOREIGN BODIES IN THE RECTUM.

Foreign bodies may enter the rectum from the alimentary canal or may be introduced through the anus. A great variety of foreign bodies have been thus introduced either by accident or design. Patients suffering from foreign body impacted in the rectum will have ineffectual attempts at defecation, with the passage of mucus, which is often blood-stained. In a case presenting these symptoms a careful exploration with the finger will enable the surgeon to ascertain the presence, the exact location, and the character of the foreign body.

Treatment.—The removal of the foreign body should be accomplished with the least possible injury to the walls of the rectum. It is well first to anæsthetize the patient, and then inject into the rectum a few ounces of olive oil. When the character and position of the foreign body have been ascertained, it may be dislodged with the finger and removed by forceps. Where the body is irregular in shape or possesses sharp edges or angles which may cause injury to the surrounding parts, retractors or a bivalve speculum should be introduced to secure free dilatation of the anus and lower portion of the rectum and facilitate removal without injury to the rectal walls. Where the foreign body consists of a large mass of inspissated material, fragmentation should be resorted to in order to secure its satisfactory removal. If the foreign body has remained in position for some time and ulceration has resulted from its presence, a solution of nitrate of silver, 10 grains to the ounce of water, should be applied to the ulcerated surface, and suppositories of iodoform should also be introduced into the rectum. Extensive ulceration of the rectum following the long-continued presence of a foreign body may be followed by stricture, and the possibility of this condition should be guarded against by judicious dilatation by the finger or bougies.

PART VII.

DISEASES OF THE NERVOUS SYSTEM.

SIMPLE CEREBRAL MENINGITIS.

BY THOMAS S. LATIMER, M. D.,
BALTIMORE.

By simple meningitis, leptomeningitis, or purulent meningitis, is usually meant inflammation of the arachnoid and pia mater. Writers distinguish an arachnitis, but as this probably never occurs apart from inflammation of the pia or dura it may be considered an unnecessary refinement. Varieties are mentioned dependent on the situation, grade, or nature of the inflammation, and whether primary or secondary, or according to the character of the exciting cause. All practical purposes are subserved by dividing simple meningitis into acute, subacute, and chronic forms, whilst considering in their appropriate places those peculiarities in each form incident to locality and origin.

All forms of meningitis have much in common, and a description of any one form is in great part a description of all; more especially is this the case in the clinical history and in the treatment; it is therefore expedient, to avoid needless repetition, which the space allotted to this article does not permit, to discuss the pathology and etiology of the different forms, and subsequently the clinical history and treatment, which are essentially the same in all.

Simple cerebral meningitis may be defined as inflammation of the arachnoid and pia mater of non-tubercular origin.

Etiology.—Simple meningitis is said to occur in utero (Guersant) and to be quite frequent in the new-born. According to Ramskill, its period of greatest frequency is prior to the second year, becoming less so from that time until after fourteen, when it again becomes more common, especially between sixteen and forty-five. Gowers places the period of greatest frequency between the ages of one and ten years, including, however the tubercular form. It is essentially a disease of early childhood, and is more common than is admitted by those who refer all basilar inflammations to a tubercular origin. In the post-mortem observations of Drs. Gee and Barlow, recorded in *St. Bartholomew's Hospital Reports* for 1878, are 6 cases of non-tubercular meningitis, and in 41 post-mortem examinations by Dr. Goodhart, in cases which he says without examination would have been set down as tuberculous, 8 were non-tubercular.

Sex may be admitted among the predisposing causes, since Parent-Duchatelet and Martinet found it to be three times as frequent in males as in females. The occupations peculiar to men and the sports of boys, involving exposure to vicissitudes of weather and to mechanical violence, may account in great part

for this difference, without assuming that there exists any liability or immunity due to sex *per se*.

Injuries to the head, extension of middle-ear inflammation or of any adjacent disease, the special cause of many specific diseases, like pneumonia, scarlatina, erysipelas, and measles, ordinary pus-producing organisms, emboli and thrombi—may all be exciting causes. An inherited or acquired predisposition is perhaps not uncommonly present, but less often than in the tubercular form. Rheumatism has been supposed to be a frequent cause, but its importance has doubtless been over-estimated. Symptoms closely simulating those of meningitis often arise in the course of acute rheumatism when post-mortem examination reveals no trace of inflammation. Trousseau absolutely denied the inflammatory nature of these cases, which he called neuroses. Two of the most characteristic symptoms of meningitis, vomiting and headache, are also commonly absent. Doubtless rheumatism is sometimes a cause of true simple meningitis, but all the symptoms may arise from hyperpyrexia alone. Suppurative endocarditis or any other septic trouble may occasion it, as in the cases following operation for imperforate anus referred to under Pathological Anatomy. Those cases arising from adjacent disease may be limited to the convexity, while those occurring in the course of acute specific diseases may affect the base also, though a preference for the convexity is recognized in all non-tubercular forms.

Pneumonia is frequently associated with simple meningitis and the pneumococcus is found in the inflammatory exudate. Huguenin states that at Zurich it is a frequent complication of pneumonia, and Chvostek found it four times in 220 cases in Vienna.

The most common cause of this affection is extension from some local adjacent disease; middle-ear inflammation is a frequent antecedent. Cases have occurred in which suppuration of the eyeball was primary, the inflammation extending along the sheath of the optic nerve. In some instances no immediately exciting cause is apparent. Fagge relates several cases occurring in Guy's Hospital in which a diseased temporal bone was found post-mortem, but the meningeal inflammation appeared to start in one instance from a blow with a bolster, and in another an attack of sunstroke preceded the cerebral symptoms about seven days. Moxon gives a prominent place to syphilis as causative of meningitis, and Fagge says 5 cases, in which it occurred without other syphilitic lesions within the calvaria, were found among the records of Guy's Hospital. This writer appears to approve the notion that the direct rays of the sun may produce simple meningitis, or that even its reflection from the pages of a book while reading is a sufficient cause; but this is scarcely credible.

Pathological Anatomy.—When death occurs in the early stage of leptomeningitis, intense hyperæmia with extreme dryness and opacity of the membranes—from distention of the lymphatic sheaths of the vessels—over the whole or part of the brain may be the only lesion. If death occur after a few days' duration, effusion of fluid admixed with cellular elements will be found on the arachnoid, in its sac and infiltrating the pia mater. Abercrombie relates a case in which it was so abundant between the dura and arachnoid as to distend the anterior fontanelle. Usually, however, the quantity of fluid exudate is not large. When life has been prolonged to the fifth or sixth day, the quantity of fluid is sensibly diminished, and a little later disappears. A membrane-like deposit of yellowish hue is found on the arachnoid; the pia in greater or less part is covered and infiltrated with "concrete pus," which is also found around the vessels and in the sulci of the convolutions (Ramskill). The nerve-

sheaths may be reddened and bathed in semi-purulent lymph, which at times is punctiform and resembles tubercular granulations. In long-standing cases this may undergo caseation or induration. The nerve-trunks may be in different stages of hyperæmia, softening, and disintegration. The dura and arachnoid may be firmly adherent, the arachnoid and pia almost always.

The ventricles may be invaded, their lining membrane inflamed, the orifices of communication occluded, and the chambers distended with serum or pus, sometimes to the extent of producing a true hydrocephalus. In rare cases they may contain false membrane. More frequently they contain a flocculent fluid of variable quantity, sometimes sufficient to distend the ventricle and compress the cortex. The subjacent brain-substance may be cedematous and softened. This condition is not always associated with unmistakable evidence of inflammation of their lining membrane; indeed, the inflammatory changes in the ventricles are rarely, if ever, well marked. In those cases where inflammation is most pronounced the effusion is seldom limited to the ventricles, but may invade the cord and escape into the brain-space. Great distention of all the ventricles may occur without inflammation, from simple occlusion of the channels of communication with the space around the brain (Gowers). Rilliet relates a case in which the convexity of one side was covered with false membrane, whilst the pia of the opposite side was simply cedematous.

Cases of pneumonic origin are usually bilateral and limited to the cortex; those extending from local foci—purulent otitis, caries, etc.—are unilateral, and may be associated with thrombi of the sinuses or with abscess (Osler). Septic cases and those associated with specific diseases are apt to be bilateral. The base is often involved in the inflammatory process. An interesting case of basilar meningitis following an operation for imperforate anus, reported by W. T. Howard, Jr., in a child of three months, is related in Osler's *Practice of Medicine*, in which the ventricles were distended with pus containing a coccus and the bacterium coli commune; the ependyma was softened and infiltrated with pus. Dr. Hilton Fagge also reports a case, occurring in Guy's Hospital, of a meningeal inflammation following six days after an operation for imperforate anus, attributed to sepsis, though the meningitis was the only evidence of pyæmia. Dr. Fagge says the presence of subdural pus may usually be taken as an evidence of extension from without, though in many cases no subdural pus is found. The pia is usually swollen and cedematous, filling the sulci; the inflammation may extend along the vessels to the cortex, which becomes infiltrated, softened, and so adherent at times that the pia cannot be removed without cortical laceration. The whole surface of the cortex may be bathed in pus or deeply infiltrated with leucocytes, and Huguenin says "suppuration of the brain-substance may reach such a point as to give rise to a diffused yellow-gray maceration visible to the naked eye" (quoted from Fagge's *Practice*). The amount of blood in the vessels may be greatly diminished from pressure of the exudate and thickening of their walls.

Symptoms.—Simple meningitis of childhood usually begins abruptly with well-marked rigors. Prodromic symptoms are much less frequent than in the tubercular form. The patient is petulant and irritable when disturbed, but inclined to apathy at other times, more especially in later stages and when the convexity is especially involved. Violent delirium with or without convulsions may be an early symptom. When convulsions occur early, they are apt to recur often during the progress of the trouble. The delirium may be quiet and the convulsions slight or absent. Pyrexia quickly supervenes, and is usually high: a temperature of 103°–105° F. is not uncommon in the first week. It is sometimes very slight, occasionally scarcely appreciable, and in the last stage

the temperature may be subnormal. The pre-mortal temperature is sometimes as high as 106°–108° F.

The pulse may be frequent and tense, usually so in the beginning, or slow and irregular, sometimes as slow as 60, 50, or 40 per minute, or just before death it may rise to 160–180 per minute. Henoch considers an intermittent pulse characteristic of meningitis. It is of more significance in childhood than in infancy, but at no time has it the diagnostic value imputed to it. The extreme variation in frequency and quality of the pulse is probably its most significant character.

Respiration is usually but little disturbed, but is sometimes sighing, may be quickened at first and subsequently irregular and slow, and toward the close the Cheyne-Stokes rhythm may be present. When the lesion is in the posterior fossa, respiration is slow, labored, accompanied by cyanosis, and may stop suddenly.

Headache is perhaps the most constant symptom, and is seldom lacking. It is often associated with great tenderness of the scalp and subjacent region, and is sometimes circumscribed, but the localization bears no constant relation to the site of the inflammation. The meninges of one side may be inflamed and the pain and tenderness be on the other; but when the pain persists in a circumscribed area it commonly indicates the site of the inflammation. Cases of simple meningitis sometimes run their entire course without pain, and when pain is present it seems to have no constant relation to the intensity or extent of the inflammation.

Hyperæsthesia of the nerves of the special senses of sight and hearing, indicated by extreme aversion to light and noise, is almost invariably present. This may be associated with acute general hyperæsthesia. The pupils are at first contracted; as the photophobia diminishes they become irregular; one may be contracted and the other dilated, or at times contracted, at times dilated; finally, both become dilated and vision is impaired or lost; optic neuritis is present in many cases, especially when the base is involved. Noises at first greatly disturb the patient. This sensitiveness to sound is at times so great that the most softly modulated speech occasions signs of petulance and distress. As the end approaches this gradually passes away, and deafness may ensue.

The intelligence is sooner or later affected; the patient is irritable and petulant when questioned or otherwise annoyed; incoherent speech and delirium are often early symptoms. Other nervous symptoms present at this time are subsultus, carphologia, inco-ordinate efforts at locomotion if this be attempted, and projectile vomiting.

The tache cérébrale is well marked, but is without diagnostic significance. Occasionally the patient emits short, sharp cries that do not always appear to be due to pain, though in older children they often seem to increase the headache.

General convulsions may occur independently of the site (Gowers), and eventually give place to coma. Rigidity of the muscles of the neck, with retraction of the head, is an early symptom of diagnostic value; it is more frequent in inflammation of the base than of the convexity. When the base is the site of the lesion, local spasm may occur in simple as well as in tubercular meningitis. Rolling up of the eyes, oscillations of the globes, strabismus, most marked when the eyes are moved, are frequently present in the first stage; later they may give place to paralysis, sometimes limited to the face or a small part of it, sometimes to a single extremity; or complete hemiplegia is present.

Vomiting is so commonly present and of such distinctive character as to possess diagnostic significance. It is projectile, unaccompanied by gastric pain or tenderness, nausea, or retching. It may persist throughout the disease, but is most characteristic in the early stage. It occurs independently of the site, but is more common in inflammation of the base. It is not present in all cases. The tongue is usually somewhat furred, but presents nothing characteristic. The bowels are constipated in a large proportion of cases, and the abdomen is retracted or boat-shaped.

Finally, all the active symptoms subside; the headache, photophobia, acoustic sensibility, general and local hyperæsthesia, and active delirium, all give place to coma and general collapse. The pupils are dilated, the pulse weak and irregular and the skin cold and clammy. Cheyne-Stokes respiration is established, the sphincters are relaxed, the fæces and urine are voided involuntarily, and death speedily ensues.

SUBACUTE LEPTOMENINGITIS is peculiar only in the relative mildness of the lesions and the slowness with which it develops. It sometimes, though rarely, succeeds to the acute form, but more commonly is subacute from the beginning. The same lesions of milder grade are present, and are due to the same exciting and predisposing causes.

Hydrocephalus is perhaps more frequent and extensive; active delirium is frequently substituted by a more quiet form and a condition of mental torpor. The patient is less irritable, the photophobia and acoustic sensibility is less, and paralyses are slower to appear. There is but little propriety, however, in recognizing a subacute form; it is merged by such insensible gradations, on the one hand, into the acute, and on the other, into the chronic form, that there is little to distinguish it.

A latent form is also described, but in the judgment of the writer it has no well-established claim to recognition, and will not therefore receive further consideration.

CHRONIC LEPTOMENINGITIS.—Chronic leptomeningitis may succeed the acute form, but is of extreme rarity except as a result of syphilis or chronic alcoholism, causes not likely to occasion it in childhood except through inheritance.

The symptoms are less clearly distinctive, and the difficulty in diagnosis therefore greater, than in the acute form; consequently it may often be overlooked and the frequency of its occurrence underestimated. If, as Goodhart has remarked, we accept cervical opisthotonos as evidence of meningitis, it may not only be very chronic, but also intermittent, and, we may add, more frequent than commonly supposed.

Its clinical history is not to be separated by sharply-drawn lines from that of the acute disease. It is essentially the same in character, but of slower development and more protracted stay, and all the more characteristic symptoms are of less intensity. An apathetic condition with headache and a disposition to vomit, a pulse at first slow, soon becoming quick and irregular, double vision, strabismus, and irregularity of pupils, may usually be found if sought for. The favorite site of chronic infantile meningitis is the posterior fossa, and the most characteristic symptoms are local and dependent on the seat of the inflammation. Drs. Gee and Barlow observed cervical opisthotonos in most cases. In some cases of rapid development it may be attended or preceded by convulsions, vomiting, pain, and fever; in others the retraction of the head is slowly induced, unattended by these phenomena. Rigidity of the limbs and epileptic convulsions may occur later, together with oscillations of the globe or strabismus, and occasionally hydrocephalus (Gowers). When the orifices of

the fourth ventricle are closed with lymph, paralyses, facial and hemiplegic, may complicate the later period. The pia is usually thickened from increase in its connective tissue; a similar condition is found in the walls of its vessels, and from them may extend to the cortex, inducing such changes as may lead to insanity and idiocy. The pia and arachnoid may be glued together, oedematous and opaque, and the sulci be filled with serum or sero-purulent fluid or oedematous membrane. The Pachionian bodies are increased in number and size.

Chronic lepto-meningitis is much more frequently associated with syphilis than is the acute form. A swollen and oedematous optic disk, or optic neuritis, may aid the diagnosis, but cannot confirm it.

One is a little at a loss to understand why the cause of the meningitis should be supposed to determine a difference in the symptoms, except in so far as these are due to associated disease. The extent, intensity, and locality of the meningeal inflammation, with the nervous susceptibility of the individual, will determine the symptoms, which will be much the same whatever the cause.

Diagnosis.—The positive indications of simple meningitis are found in the symptoms already mentioned, though they may any or all of them occur without meningeal lesion of any kind whatever. The general cerebral symptoms are valuable according to their degree and combination, rather than by their mere presence. "The significance of the headache depends on its intensity; of the delirium, on its coexistence with headache; of vomiting, on its causeless character and persistence; of general convulsions, on their association with other symptoms; of infrequency of pulse, on its combination with pyrexia that usually accelerates the heart" (Gowers). It is not to be distinguished by its symptoms from tubercular meningitis, though in general it may be said to be more frequently dependent on some pre-existing local disease, to be more abrupt in its invasion and rapid in its progress in acute cases, and to be more frequently associated with active delirium. It is probably more dependent on some local lesion or association with specific disease than is the tubercular form. In the latter the presence of the tubercle bacilli or of septic materials from degenerating tubercles, with peculiar susceptibility, is alone sufficient for its development. The presence, therefore, of tubercle in other organs, the detection of tubercle bacilli, and a tubercular family history are of greater value in the differential diagnosis than any supposed difference in symptoms directly due to the meningitis. Though clear evidence of tubercle elsewhere may be wanting, slow invasion, early childhood, and the absence of distinct local cause make for a tuberculous origin. When the base alone is the site of the inflammation, the probabilities are strongly in favor of the tubercular form. Inflammation of the middle ear or labyrinth, with or without suppuration, may give rise to symptoms that cannot be distinguished, except by their duration, from meningitis. The detection of an otitis, therefore, may lead us to believe in the existence of meningitis originating from it, or to hope that the symptoms are solely due to it and will end in recovery under proper treatment. And in cases that recover under such circumstances, the diagnosis must remain permanently in doubt, since many cases of simple meningitis have been thought to recover.

It is also not altogether unlikely that the characteristic symptoms may arise as a reflex result of lesions of the most varied character in remote parts of the body. From the cerebral form of pneumonia simple meningitis may be distinguished by the physical signs of the former and the detection of the pneumococcus. But it must be remembered that although pneumonia may exist without meningitis, with analogous cerebral symptoms, yet pneumonitis and true meningitis may coexist and be due to the same cause. I know of no way to

distinguish cerebral symptoms occurring in pneumonia without meningitis and those occurring under like circumstances with it, except by their duration; and even this in many cases is the same, since pneumonia with marked cerebral symptoms often runs a speedily fatal course. Perhaps, instead of trying to differentiate them, it would be best to consider both as local expressions of the same constitutional state.

Pyæmia may present symptoms closely resembling meningitis, especially when associated with thrombus of the lateral sinus and jugular vein, as in a case reported by Dr. Frederick Taylor. Dr. Wilson Fox also relates a similar case, and Dr. Andrew two instances of pyæmia with cerebral symptoms not distinguishable from meningitis; both of these recovered, however, so it cannot be said they were not cases of true meningitis, unless it be assumed that acute simple meningitis never recovers.

Those cases of typhoid fever likely to occasion difficulty in diagnosis are characterized by the predominance of cerebral symptoms and the absence or slight nature of those peculiar to the alimentary canal; but in typhoid fever headache precedes delirium, usually ceases with its advent, and is sufficiently accounted for by the pyrexia—not so in leptomeningitis. Photophobia and auditory hypersensibility may occur in either, but they are far more acute in meningitis. In typhoid fever vomiting seldom has the distinctive cerebral character, and rigidity of the neck and local paralyses seldom occur. The invasion of typhoid fever is rarely so abrupt; the pulse is not so irregular.

Prognosis.—In all cases of leptomeningitis but little hope can be reasonably entertained of recovery when no error in diagnosis has been made; but errors of this kind happen in the experience of the most astute and well-informed physicians. Moreover, cases apparently free from doubt have recovered in sufficient number to warrant hope, but hope only, for nothing in the condition of the patient serves as a reasonable basis for expectation of recovery. The cases which do best are those having their origin in injuries, necrosis, caries, suppurative otitis, and other removable causes, and those that arise in the progress of syphilis. The majority even of these will terminate fatally after the inflammation is well established, but much may be done, by the early removal or correction of such causes, to prevent the establishment of meningitis. Those in which the indications are that both the convexity and base are affected run a rapidly fatal course. Patients in whom no reasonable cause exists may be expected to succumb, more especially if the pulse soon becomes irregular and weak, accompanied by nausea, with convulsive seizures followed by profound hebetude.

When light and noise no longer disturb; when the pupils become persistently dilated; the skin cold, pale, and bathed in perspiration; when involuntary evacuations occur; paralysis local or general becomes established, and coma or a semicomatose condition supervenes,—all hope may be abandoned.

Treatment.—The treatment in simple cerebral meningitis and in simple cerebro-spinal meningitis is essentially the same. A much larger proportion of cerebral cases are due to local conditions that may be treated by surgical measures, and whenever they do arise from removable causes surgical treatment should be instituted without delay. Suppurative otitis, with or without necrosis or caries of the temporal bone, is so often causally related to meningeal inflammation that these lesions should always receive efficient attention before the induction of the graver evil. It has happened to the writer to witness two cases of supposed leptomeningitis in adults, with fatal terminations, supervening on chronic suppurative otitis that had followed scarlatina many years before. Had the aural trouble been efficiently treated, the meningeal inflammation

would in all probability have been averted. Doubtless many similar cases have existed, and, in view of the great fatality of the secondary affection and the impunity with which surgeons of the present day invade the meninges, and even the substance of the brain, they should in future disappear from our records. All cases of injury to the skull that carry with them even a reasonable suspicion of injury to the meninges or brain should, in the judgment of the writer, be trephined, bone-fragments elevated or extracted, blood-clots removed though the membranes have to be incised for that purpose, and all the parts thoroughly cleansed. Analogous procedures are no less imperatively called for in diseased conditions than after injury.

Cases of syphilitic origin should receive the specific treatment proper to that disease, with a not unreasonable hope of recovery if the treatment be begun early.

Apart from these special indications for treatment, there is but little to be expected from any means at our disposal beyond the alleviation of suffering. Drugs appear to exert no influence on the course of the disease, and it may be doubted, even in those cases of supposed leptomeningitis that have recovered, whether the remedies administered have contributed to this result.

Bleeding, local or general, and blisters are still strongly advocated by German writers and by many others. Apart from relief of hyperæmia of the cerebral vessels, one sees but little benefit to be derived from them, and it would seem that this might be better accomplished by such remedies as amyl nitrite, which increase the vascular area and so lower blood-pressure without the same impairment of strength as follows bloodletting. Mercury and the iodide of potassium have been warmly and ably advocated as efficient therapeutic agents in this disease, but they so often appear to be entirely without effect that the writer is sceptical of their value except in cases of syphilitic origin. Nevertheless, excellent results of treatment with these agents have been reported by most competent observers. Ramskill in Reynolds's *System of Medicine* thus summarizes the treatment: "It resolves itself into three great remedial measures: first, bloodletting; second, hard purging; third, applications of cold water or ice to the head."

Abercrombie's cases also give strong support to the efficiency of these measures. Case 69, aged eleven, after an illness of five or six days was in a comatose condition, notwithstanding free purging, blistering, and the use of mercury to salivation; was immediately relieved and made a good recovery after one bleeding from the arm. Case 72, aged twenty-one years, was reduced to a condition of stupor from which he could scarcely be roused, and continued in this way for eight or ten days notwithstanding repeated bleeding, blisters, and cold applications. But, after taking full doses of castor oil every three hours until purgation was induced, he was on the same evening relieved and made a good recovery (quoted in Fagge's *Practice*).

A brisk purge in the beginning and from time to time during the progress of the trouble will do much to alleviate suffering, and at times seems to have a decidedly beneficial effect. Cases, not a few, are recorded, especially by the earlier writers, which seem to date improvement, rapidly progressing to recovery, from such measures as free catharsis, bleeding, local or general, blisters applied to head or back of neck. Whilst it is difficult wholly to discredit such statements or to deny to the agents employed the remedial potency claimed for them, yet it is equally difficult to conceive how with such means such ends could be attained. When remedies of this class are serviceable at all, it must be in the early stage before inflammatory exudation, infiltration, or degenerative changes have occurred to any notable extent; and one cannot wholly

shake off the doubt that they were cases of erroneous diagnosis, or at least belong to that rare class of cases that would have recovered without medical interference.

An entirely different class of remedies is found in those drugs of anodyne and hypnotic properties which allay vomiting, soothe pain, subdue or lessen active delirium, procure sleep, and contribute in many ways to make tolerable for patients and friends the last days of those for whom in a large majority of cases we can hope to do no more. And, in the opinion of the writer, relief in these particulars is the extent of the power of drugs to be useful in this disease. First rank in this group of remedies may still be boldly claimed for opium and its derivatives, and especially for morphine, which, because of the small dose required, the facility with which it may be administered hypodermatically, and its almost uniform strength and efficiency, takes precedence of all other drugs. The bromides of sodium and potassium, antipyrin, antifebrin, sulphonal, and many other similar remedies are at times of great usefulness. The occasional use of chloroform by inhalation when convulsions occur gives prompt relief to some of the most distressing symptoms, and is, I believe, as free from danger as any other remedy when judiciously employed; but nothing can be so confidently relied on to relieve pain, to procure sleep, to quiet delirium, and to arrest vomiting as morphine; and this it does at as little cost to a feeble heart as any other drug that may be used; nor do I think is the tendency to coma materially, if at all, increased by its judicious administration. But more valuable than any of the remedies yet mentioned is repose of body and mind as perfect as may be had by the mere exclusion of disturbing causes. The room should be darkened; no one not indispensable to the comfort of the patient should be admitted; no loud talking or other noises should be allowed within hearing; and no needless questioning by anxious friends. In cerebral cases the head should be shaved as soon as the nature of the trouble is clear. Ice or ice-cold water should be almost continuously applied to the head and—in cerebro-spinal cases—to the back. It should be begun early and continued steadily, and in cases of active delirium this may be supplemented by iced applications to the large arteries—brachials and carotids.

When coma appears, all depressing remedies should cease, although occasional recoveries are recorded even in this stage, as in Abercrombie's cases, already quoted, and in Sir Thomas Watson's case of recovery on the application of a blister to the entire shaven scalp after the appearance of coma. It is certainly more reasonable to expect good from the judicious use of stimulants in this stage or at any time when the heart-beat is feeble or intermittent. Throughout the disease, as far as practicable, the strength of the patient should be maintained with the most nutritious diet.

